DISSERTATION ON

‘THE PREVALENT RISK FACTORS OF ACUTE CORONARY SYNDROME IN FEMALES’

DISSERTATION SUBMITTED TO

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

IN PARTIAL FULFILMENT OF THE REGULATIONS FOR THE AWARD OF THE

DEGREE OF M.D. - GENERAL MEDICINE- BRANCH – I

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004.

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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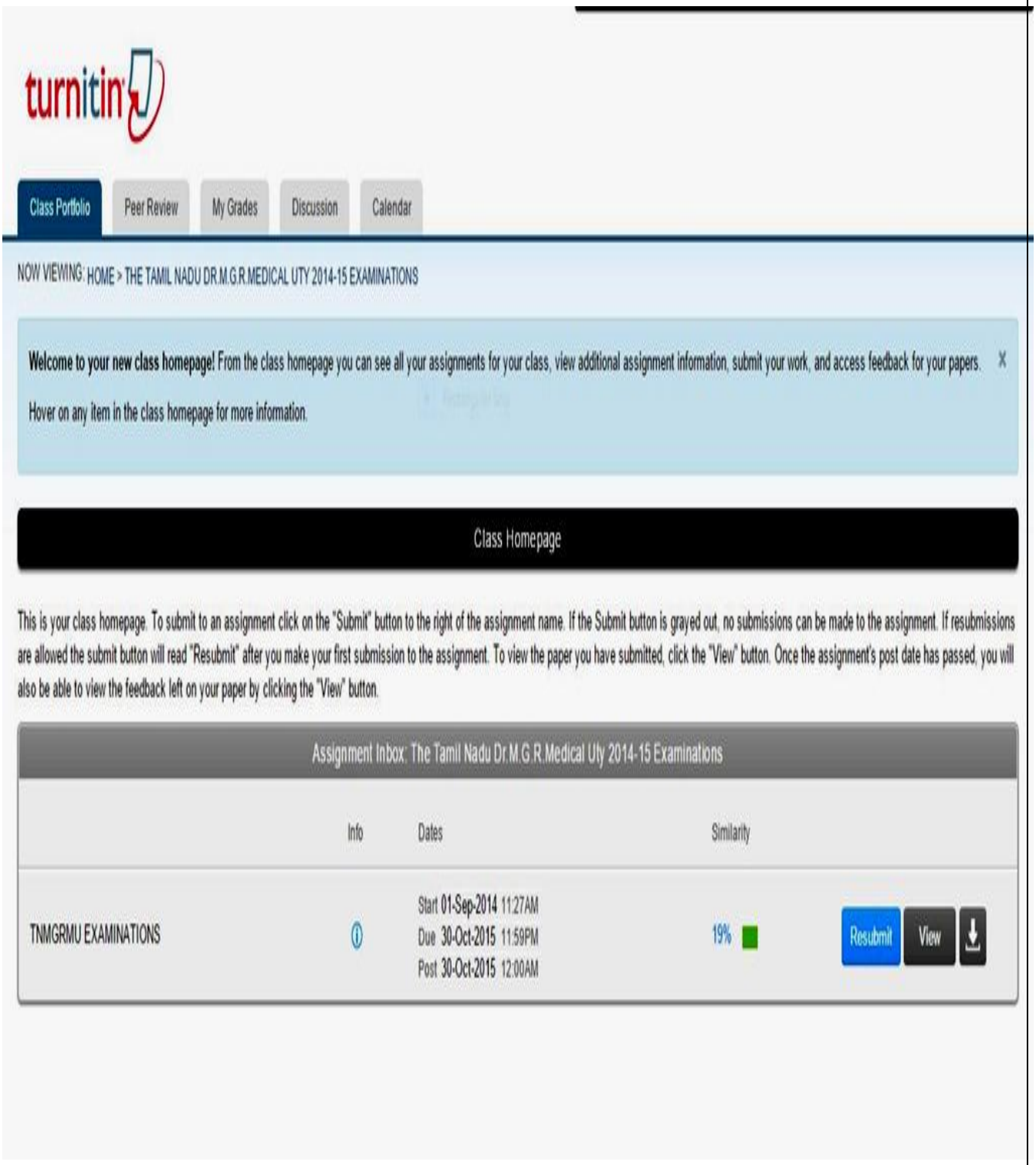
M.D. - GENERAL MEDICINE - BRANCH - I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DECLARATION

I, **DR SOBIN E. JOSEPH**, solemnly declare that the dissertation titled **THE PREVALENT RISK FACTORS OF ACUTE CORONARY SYNDROME IN FEMALES** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during **2014 DECEMBER TO 2015 MAY** under the guidance and supervision of **Prof.Dr.C.GANESAN, M.D.,** Unit Chief, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to Dr. M.G.R Medical University, Tamil Nadu towards partial fulfillment of requirement for the award of **M.D. degree (Branch -I) in General Medicine**.

Place: THANJAVUR

Date:

DR SOBIN E. JOSEPH

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Last but not the least, I also thank all my patients for their cooperation and patience, without whom this study would not have been completed.

A special mention to my family and friends for their unfailing support.

CONTENTS

From

DR.SOBIN E. JOSEPH
MD (GENERAL MEDICINE) POST GRADUATE,
Department of General Medicine,
Thanjavur Medical College & Hospital,
Thanjavur .

To

THE CHAIRMAN,
INSTITUTIONAL ETHICAL COMMITTEE (IEC),
Thanjavur Medical College & Hospital,
Thanjavur.

Through proper channel,

Respected Sir/Madam,

SUB: Request for approval from the Institutional Ethical Committee to conduct a study on **THE PREVALENT RISK FACTORS OF ACUTE CORONARY SYNDROME IN FEMALES**

I have proposed a dissertation work on **THE PREVALENT RISK FACTORS OF ACUTE CORONARY SYNDROME IN FEMALES**. As this study involves human beings, I request an approval from the Ethical committee.

I am enclosing the details of the study work and submitting the following undertaking

1. I will get the detailed informed written consent from the patients/participants and maintain confidentiality.
2. I will carry out the work without being detrimental to regular activities as well as without extra expenditure to the Institution, Government and the participants.
3. I will inform the committee in case of any change in the study procedure, site and investigations done.
4. I will not deviate from area of work for which I have applied ethical clearance.
5. I will inform IEC immediately in case of any adverse events or reactions.
6. I will abide by rules and regulations of the institution.
7. I will complete the work within the specified period I applied for, if any extension of time is required, I shall apply for permission again and do the work.
8. I will submit the summary of the work to the Ethical Committee on completion of the work.
9. I will not claim funds from the institution during my work or on completion.
10. I understand that the members of the IEC have the right to monitor the work.

Thanking you,

Date: Yours Sincerely,

Station DR SOBIN E.JOSEPH

ENCLOSURE: Copies of study proposal.

GUIDE:

PROF. DR.C.GANESAN MD ,
PROFESSOR OF MEDICINE
M3 UNIT CHIEF,
DEPARTMENT OF GENERAL MEDICINE,
THANJAVUR MEDICAL COLLEGE & HOSPITAL,
THANJAVUR

CHIEF CO-ORDINATOR:

Prof. Dr. K. NAGARAJAN MD
THE PROFESSOR AND HOD,
DEPARTMENT OF GENERAL MEDICINE,
THANJAVUR MEDICAL COLLEGE &
HOSPITAL,
THANJAVUR

Remarks of the Guide:

The work done by DR SOBIN E.JOSEPH on **THE PREVALENT RISK FACTORS OF ACUTE CORONARY SYNDROME IN FEMALES** is under my supervision and I assure that this candidate will abide by the rules of the Ethical Committee.

GUIDE:

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PROFESSOR OF MEDICINE,
M3 UNIT CHIEF
DEPARTMENT OF GENERAL MEDICINE,
THANJAVUR MEDICAL COLLEGE & HOSPITAL,
THANJAVUR

RECOMMENDATION FROM THE HEAD OF THE DEPARTMENT:

The dissertation titled **THE PREVALENT RISK FACTORS OF ACUTE CORONARY SYNDROME IN FEMALES** is being done according to the regulations of the Ethical committee and I recommend it for its acceptance.

Prof. Dr. K.NAGARAJAN M.D. ,
THE PROFESSOR AND HOD OF MEDICINE,
DEPARTMENT OF GENERAL MEDICINE,
THANJAVUR MEDICAL COLLEGE &
HOSPITAL,
THANJAVUR

THE PREVALENT RISK FACTORS OF ACUTE CORONARY SYNDROME IN FEMALES

INVESTIGATOR : **DR SOBIN E.JOSEPH**
MD (GENERAL MEDICINE) PG

STUDY PERIOD : **2014 DECEMBER TO 2015 MAY**

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THE PROFESSOR AND HOD OF MEDICINE,
DEPARTMENT OF GENERAL MEDICINE,
THANJAVUR MEDICAL COLLEGE & HOSPITAL,
THANJAVUR

Details of the Study submitted by individual desirous of clearance from Institutional Ethical Committee

TITLE:
THE PREVALENT RISK FACTORS OF ACUTE CORONARY SYNDROME IN FEMALES

AIMS AND OBJECTIVE :

The study is designed to evaluate the frequency of prevalent risk factors of ACS in females admitted in the medicine casualty of Thanjavur Medical College

STUDY PLACE : Thanjavur Medical College & Hospital,
Thanjavur – 613004.

COLLABORATING DEPARTMENT: -

STUDY DESIGN **:** CROSS SECTIONAL STUDY

STUDY PERIOD **:** **2014 DECEMBER TO 2015 MAY**

ETHICAL CLEARANCE **:** **Applied for Institutional clearance**

INCLUSION CRITERIA **:** **ACS IN FEMALE PATIENTS DIAGNOSED BY;**
1. **Symptoms s/o ACS with definitive ECG changes**
2. **Or raised cardiac markers**

EXCLUSION CRITERIA **:** **chest pain with normal ECG / normal cardiac enzymes**

INVESTIGATION **:**
1.RANDOM BLOOD SUGAR
2.SERUM UREA AND CREATININE
3.FASTING LIPD PROFILE
4.SR.ELECTROLYTES
5.ECG
6.TROPONIN T

DATA COLLECTION **:** Clinical

BENEFIT TO THE COMMUNITY:
THIS STUDY AIMS AT BENEFITTING THE COMMUNITY BY UNDERSTANDING THE FREQUENCY AND IMPACT OF PREVALENT RISK FACTORS OF ACS IN FEMALES AND THEREBY,ESTABLISHNG THE IMPORTANCE OF PRIMORDIAL PREVENTION.

FINANCIAL SUPPORT : NIL

PRINCIPAL INVESTIGATOR : **DR.SOBIN E.JOSEPH**
MD (GENERAL MEDICINE) P.G

GUIDE : **PROF. DR C.GANESAN M.D. ,**
PROFESSOR OF MEDICINE
M3 UNIT CHIEF,
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THANJAVUR

CHIEF CO-ORDINATOR : **Prof. Dr. K. NAGARAJAN M.D. ,**
THE PROFESSOR AND HOD OF MEDICINE,
DEPARTMENT OF GENERAL MEDICINE,
TMCH
THANJAVUR

PROFESSIONAL SUPPORT : ASSOCIATE & ASSISTANT PROFESSORS ,
THANJAVUR MEDICAL COLLEGE.

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR SOBIN E. JOSEPH,** Post graduate in the Department of General Medicine ,Thanjavur Medical College & Hospital, Thanjavur – 613004 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations

Place :
Date : Signature of participant

INFORMATION SHEET

We are conducting a cross sectional study on THE PREVALENT RISK FACTORS OF ACS IN FEMALES

in the Department of General Medicine , Thanjavur Medical College & Hospital, Thanjavur – 613004.

- At the time of announcing the results and suggestions, name and identity of the patients will be confidential.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator **Signature of participant**

Date:

DETAILS OF THE STUDY SUBMITTED BY INDIVIDUAL DESIROUS OF CLEARANCE FROM INSTITUTIONAL ETHICAL COMMITTEE.

Title	THE PREVALENT RISK FACTORS OF ACUTE CORONARY SYNDROME IN FEMALES
Aims And Objective	**The study is designed to determine the impact of prevalent risk factors of ACS in females**
Design of the study	Cross sectional study
Period of the study	December 2014 to may 2015
Ethical clearance	Applied for Ethical committee clearance
Consent	An informed consent will be obtained
Materials & Methods/ Selection of study subjects	Cases of female patients admitted in the medicine casualty of Thanjavur Medical College & Hospital , who satisfy the inclusion criteria are studied with history, clinical examination, ECG , Random Blood Sugar ,Serum Urea, Creatinine, Fasting lipid profile and Troponin T.
Inclusion criteria	**ACS IN FEMALE PATIENTS DIAGNOSED BY;** • **Symptoms s/o ACS with definitive ECG changes** • **Or raised cardiac markers**
Exclusion criteria	**1.CHEST PAIN WITH NORMAL ECG /NORMAL CARDIAC MARKERS**
Analysis	The collected data will be analyzed using statistical package
Conflict of Interest	Nil
Financial Support	Nil
Participant's Principal Investigator	**DR.SOBIN E.JOSEPH** **MD (GENERAL MEDICINE) P.G**
Supervision and Guide	**PROF. DR.C. GANESAN MD. ,** PROFESSOR OF MEDICINE M3 UNIT CHIEF, DEPARTMENT OF GENERAL MEDICINE, THANJAVUR MEDICAL COLLEGE & HOSPITAL THANJAVUR

INTRODUCTION

Cardiovascular disease has emerged as one of the leading cause of death in women, resulting in the death of one in three women, irrespective of race and ethnicity.

A growing body of research based on gender influence in cardiovascular disease is elucidating the differences between men and women. Be it clinical presentation or the response to treatment, the profile variation between the two sexes are profound.

Women with ACS more commonly present with non cardiac chest pain, have negative cardiac biomarkers and are usually referred for treatment late. Despite of a lower incidence of STEMI in women than men, the morbidity and mortality rates are significantly higher in women. This can be partly attributed to the delay in treatment due to the lack of awareness and less aggressive treatment being offered to women which is evidenced by the low rates of PCI in women.

The influence of sex-age interaction on mortality rates in cardiovascular disease have long been under study. Various researches have proven that younger women had a higher in patient mortality than men of the same age. As compared to young men, the prevalence of DM, CHF and late presentation was higher in young women. In addition to this, they had a higher chance of developing complications.

Most of our knowledge on MI and treatment profile has been developed from studies which focused mainly on men. In the present scenario, it is important to understand

the subtle differences in the development and progression of disease in men and women. Most of these differences can be attributed to age and the prevalence of co morbidities. Therein lay the supreme need to identify those risk factors which predispose women to cardiovascular disease and implement this knowledge in our day to day out patient practice.

AIMS AND OBJECTIVES

- The study is designed to evaluate the frequency of prevalent risk factors of ACS in females admitted in the medicine casualty of Thanjavur Medical College

REVIEW OF LITERATURE

ANATOMY

The borders of the heart:

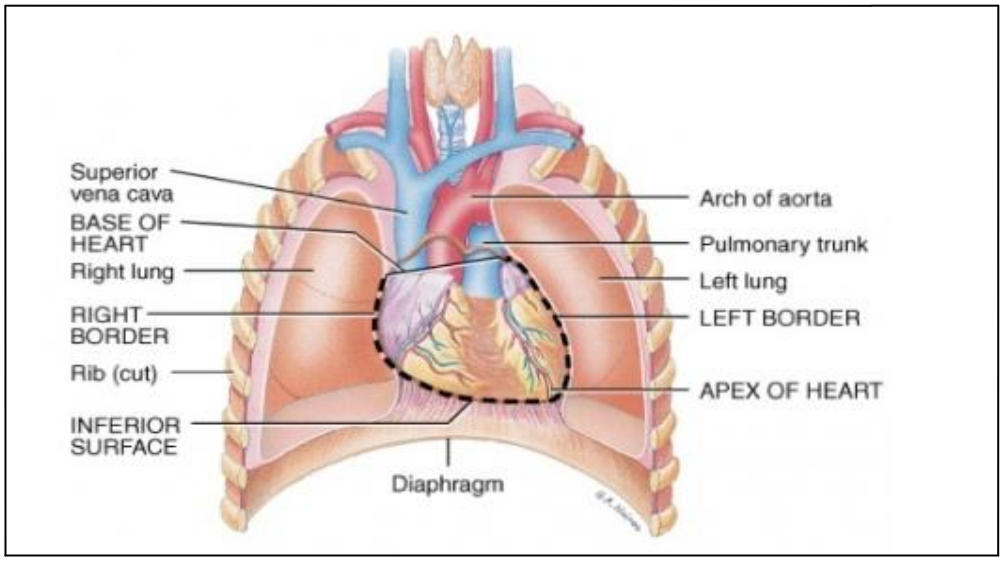
Right border of the heart corresponds to right atrium and is in line with the SVC and IVC.

Left border of the heart is comprised mainly by the left atrium. The left auricular appendages also contribute a little to it.

The right ventricle and a portion of the left ventricle form the inferior border.

Four veins (right & left pulmonary veins) open into the left atrium.

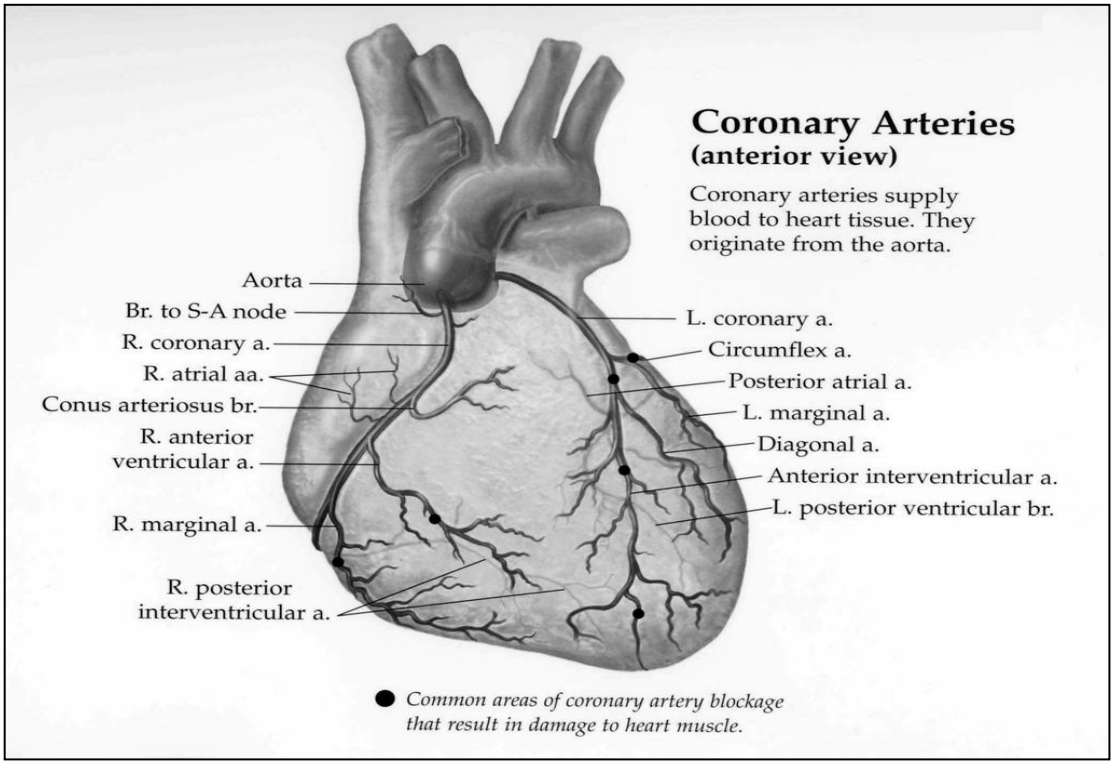
Diagram no: 1) borders of the heart



Blood supply of the heart

The major blood supply is by the right & left coronary arteries which are branches of the ascending aorta.

Diagram no: 3



RCA lies in the coronary sulcus & anastomose with left circumflex artery on the posterior aspect

Branches: large - marginal, posterior, inter ventricular

Small- nodal, R .atrial , infundibular , terminal

Areas of distribution : right atrium, right ventricle except area adjoining anterior inter ventricular groove ,posterior part of inter ventricular septum, conducting system of heart except left branch of AV bundle .

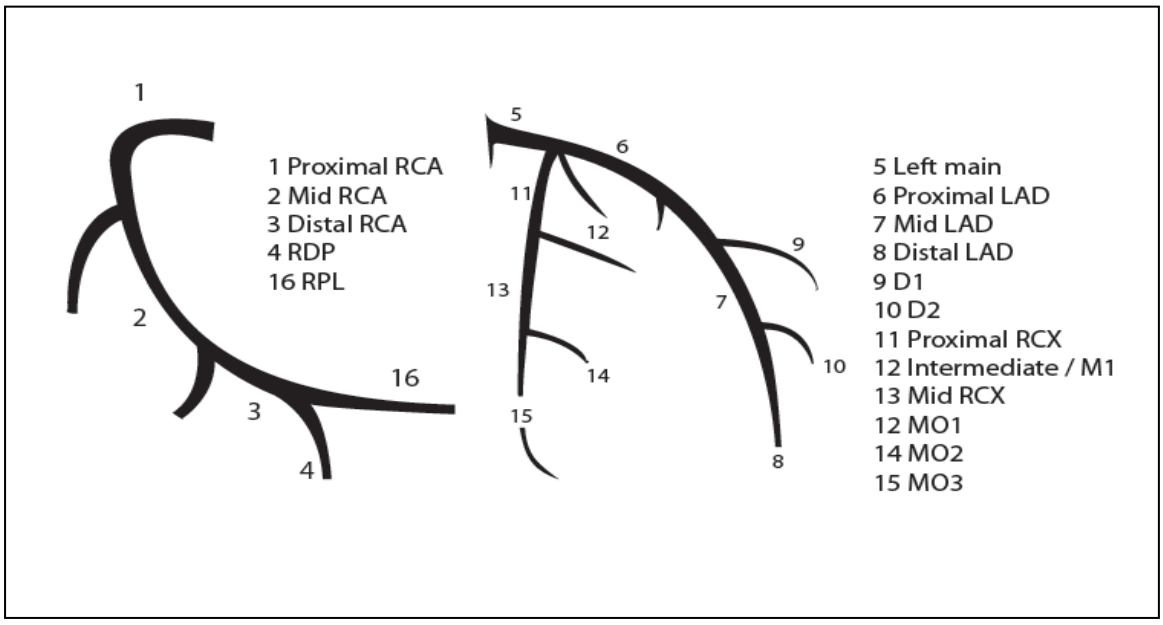
LCA

Branches: large – diagonal, anterior inter ventricuar

Small- left atrial, pulmonary, terminal

Areas of distribution: left atrium, left ventricle except area adjoining posterior inter ventricular groove , anterior part of inter ventricular septum ,left branch of AV bundle, small part of right ventricle adjoining anterior inter ventricular groove

Diagram no: 4



The dominant system of blood supply is the one which gives off the posterior inter ventricular branch. Most commonly it is the left coronary artery in 70 - 75% of the people.

In fetal life, the coronary arteries are supplemented by a rich supply of collaterals. In contrast, adult coronary arteries are end arteries, collaterals develop during chronic hypoxia.

MYOCARDIAL INFARCTION

Myocardial infarction refers to ischemic necrosis of the heart muscles (myocardium).There is a mismatch between the oxygen demand and blood supply to the area

In 2001, the American College of Cardiology issued a recommendation for the diagnosis of Acute MI replacing the old version of World Health Organization definition.

The rise and fall of cardiac markers (CK-MB, Troponin) in addition to:

- Typical symptoms of MI like angina, palpitations, diaphoresis
- ST depression/ elevation in the electrocardiogram
- New onset pathological deep Q waves

Autopsy findings suggestive of MI are also considered diagnostic. Additionally STEMI can be diagnosed in a patient presenting with typical clinical symptoms of MI along with new onset left bundle branch block. However distinction between UA and NSTEMI may prove difficult in the first few hours within the onset of the event since detectable elevations in cardiac makers may not be apparent during that time period. So serial measurements are required.

Revised Definition of Myocardial Infarction (MI)

A. Criteria for Acute, Evolving, or Recent MI

1. Typical rise and/or fall of biochemical markers of myocardial necrosis with at least one of the following

A) Ischemic symptoms

B) Development of pathological Q waves in the ECG

C) ECG changes indicative of ischemia (ST segment elevation or depression)

D) Imaging evidence of new loss of viable myocardium or new regional wall Motion abnormality

2. Pathological findings of an acute myocardial infarction. [1]

(Either of the above criteria satisfies the diagnosis for acute, evolving or recent MI)

B. Criteria for Healing or Healed Myocardial Infarction:

1. Development of new pathological Q waves in serial ECGs. The patient may or may not remember previous symptoms. Biochemical markers of myocardial necrosis may have normalized depending on the length of time that has passed since the infarction developed.

2. Pathological findings of a healed or healing infarction.

(Any one of the above criteria satisfies the diagnosis for healing or healed MI)

Ischemic heart disease is caused either by obstruction in the blood flow or increase in oxygen demand caused by cardiac chamber hypertrophy.

The commonest cause is atherosclerosis which can cause obstruction or narrowing of a coronary artery sufficient to reduce blood flow and inadequate perfusion to that area.

Despite the above data, studies have shown declining mortality in IHD which can be attributed to the improvement in health facilities and increasing awareness among the general public. [2]

The modifiable risk factors for myocardial infarction include a sedentary lifestyle ,high calorie diet and obesity .Familial hyperlipidemia , insulin resistance ,type 2 DM, hypertension are important risk factors for MI. Highest susceptibility is seen among the southeast Asian countries

Pathophysiology

Causes of Acute MI:

3) Arteritis 4) Intimal proliferative diseases- • Homocystinemia • Fabrys disease • Amyloidosis • Aortic Dissection	• Juvenile intimal sclerosis • Oral contraceptive pills • Mucopolysaccharidoses 1) Luminal narrowing • prinzmetals angina ; spasm without thrombus • Nitroglycerin withdrawal

Coronary artery Embolism • Acute bacterial endocarditis • Libman sachs and marantic endocarditis • left ventricular stasis/ thrombus • Cardiac tumours • Post procedures like CABG and PTCA **Congenital anomalies** • ALCAPA	**Myocardial oxygen demand-supply mismatch** • Aortic stenosis, • Cardiac chamber hypertrophy • Severe anemia • Pagets disease • Cardiogenic shock

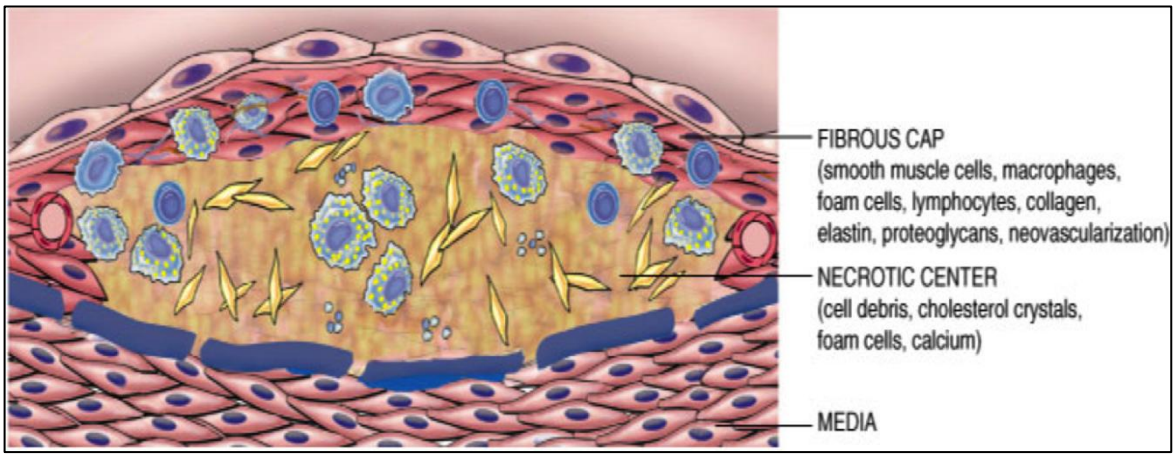
Pathophysiology of atherosclerosis

Since it is the most important cause, it is essential to describe atherosclerosis in detail

The pathognomonic feature of atherosclerosis is the atheroma or atherosclerotic plaque. Histopathology shows

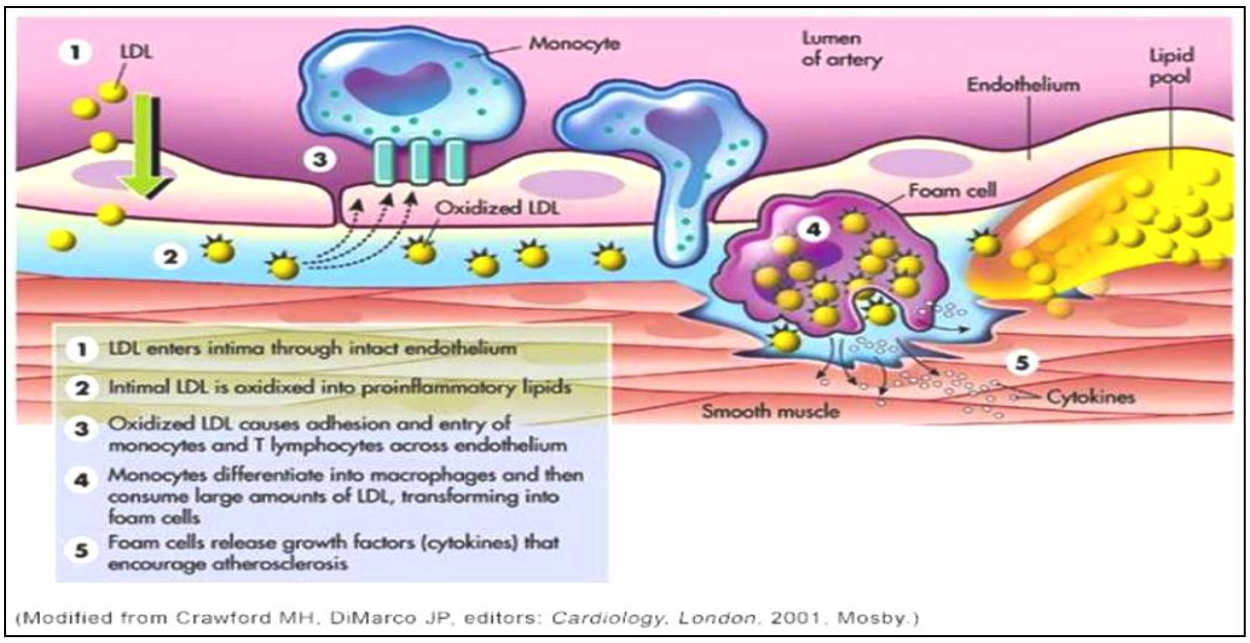
- Fibrotic cap
- Cellular area around cap having macrophages ,smooth muscles and T lympocytes
- Necrotic core

Diagram no: 5 image of an atheromatous plaque



Chronic endothelial injury results in increased permeability, leukocyte adhesion and thrombotic potential. This is associated with accumulation of lipoproteins and its oxidation in the vessel walls. This further leads to more accumulation of monocytes which adhere to the vessel wall and transform into macrophages and foam cells inside the intima along with adhesion of platelets.

Diagram no 6: pathophysiology of atheromatous plaque



The activated platelets release factors causing migration of smooth muscles from media to the intima and their proliferation along with deposition of proteoglycans and collagen. This results in enhanced accumulation of lipids. The metallo proteinases causes thinning and erosion of the fibrous cap, resulting in luminal narrowing/ occlusion. [3]

PLAQUE STABILITY

There are 3 factors which determine plaque stability

- Mechanical stress
- Inflammatory triggers
- Weakening of the fibrotic cap(which can be caused by different factors)

Diagram no 7

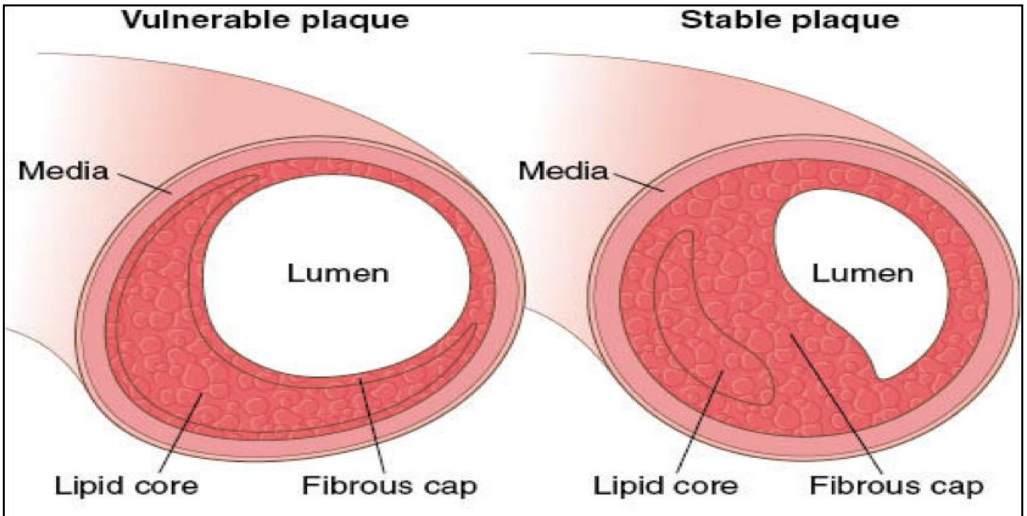


FIGURE 11–16 Schematic comparing vulnerable and stable atherosclerotic plaque. Whereas stable plaques have densely collagenous and thickened fibrous caps with minimal inflammation and negligible underlying atheromatous core, vulnerable plaques (prone to rupture) are characterized by thin fibrous caps, large lipid cores, and increased inflammation.

Mechanical stress

Stress felt by the atheromatous plaque is dependent on several factors. One is the thickness of the cap. A relative thin cap is seen in moderate stenosis as compared to severe stenosis, so moderate stenosis has to endure more stress. The lipid pool overlying the cap is also important. Along with the quantity of lipid pool, the nature of accumulated lipids is also important. Softer plaques withstand mechanical stress poorly. The softness is determined by the ratio of cholesterol esters to monohydrate cholesterol as well as the size of the necrotic lipid rich core of a plaque relative to the size and thickness of the cap.[4]

Weakening of the cap

- Reduced collagen synthesis by smooth muscle cells in the vulnerable region of plaques

  The collagenous framework of the plaque's fibrotic cap plays a crucial role in maintaining the strength of the cap. The immune and inflammatory factors disturb the framework by the secretion of IFN-gamma which inhibits collagen synthesis.

- Increased collagen degradation

Normal human arteries do not have MMPs, which are capable of breaking down interstitial collagen. However, certain cells in the atheroma secrete MMPs 1, 8 and 13 and they are capable of degrading collagen in situ

- Enhanced smooth muscle death

As mentioned earlier, inflammatory mediators like interferon gamma secreted by activated T cells inhibit collagen synthesis and smooth muscle replication. Along with this, certain pro-inflammatory mediators like interleukin 1 beta and tumor necrosis factor can promote apoptosis of smooth muscle cells. These pro-inflammatory mediators can also cause programmed cell death of endothelial cells and lysis of basement membrane (type IV collagen by collagenases especially MMP 2)

These are potential mechanisms of superficial erosions being formed on plaques and thereby stimulate platelet adhesion.

- Inflammatory triggers

Active inflammation plays a major role in the chronic pathogenesis of atherosclerosis .Many patients with severe and extensive atherosclerosis remain stable for many years, while some patients present with severe and acute complications of ACS as the first manifestation. This exposes our relative lack of knowledge about the inflammatory triggers that bring about acute complications of ACS.

A large part of vessel wall is composed by the media and adventitia and perfused by vasa vasorum. These vessels undergo proliferation in atheromatous lesions. Angiogenic factors like vascular endothelial growth factor, transforming growth factor etc are secreted at the site of lesions in response to local stress, intramural hypoxia, inflammation and apoptosis .These factors cause proliferation and sprouting of new vessels in and around the atheromatous plaques

Initially, these neo-vessels serve as a compensatory mechanism to counteract local hypoxia and helps restoring oxygen availability and resolving inflammation. Recent works have revealed that these immature neo-vessels which lack basement membrane are a potential precipitating factor for ACS. It causes intra plaque hemorrhage , leading to rapid plaque expansion and fuels inflammation.

Activated inflammatory cells secrete pro inflammatory cytokines which promote neo-vascularisation, leakage of blood and generation of free Hb. If this free Hb is not rapidly bound by haptoglobin and subsequently cleared by CD163 positive macrophages ,a sudden surge of oxidative stress occurs. Reactive oxygen species which overwhelm the local radical scavenging mechanisms are produced. This leads to further leukocyte activation and the vicious cycle continues.

Research groups like *Yunoki et al* have made an important observation that neutrophils were specific for unstable plaques. Telomerase enzyme in neutrophils of unstable show transient activation; this is not seen in stable plaques. Similarly differences in T lymphocytes have also been reported.

The greatest challenge of the future would be to determine those specific triggers which can destabilize a plaque and initiate the dreaded complications of ACS.

RISK FACTORS FOR ATHEROSCLEROSIS

NONMODIFIABLE	MODIFIABLE
1) Elderly (men >45 & women > 55) 2) Male gender 3) Family history of early onset CAD (at age less than 55 years) 4) Genetic abnormalities	1) Hyperlipidemia ❖ Total cholesterol > 150mg% ❖ Triglycerides > 150 mgs% ❖ LDL cholesterol > 100 mg% ❖ APO – b lipoproteins >100 mg % ❖ HDL cholesterol < 40 mg% males, < 50 mg% females. 2) Hypertension 3) Cigarette smoking 4) Diabetes Mellitus 5) C-reactive protein 6) BMI > 22 7) Homocysteine > 10 micro mol/lit

Stages of atherosclerosis

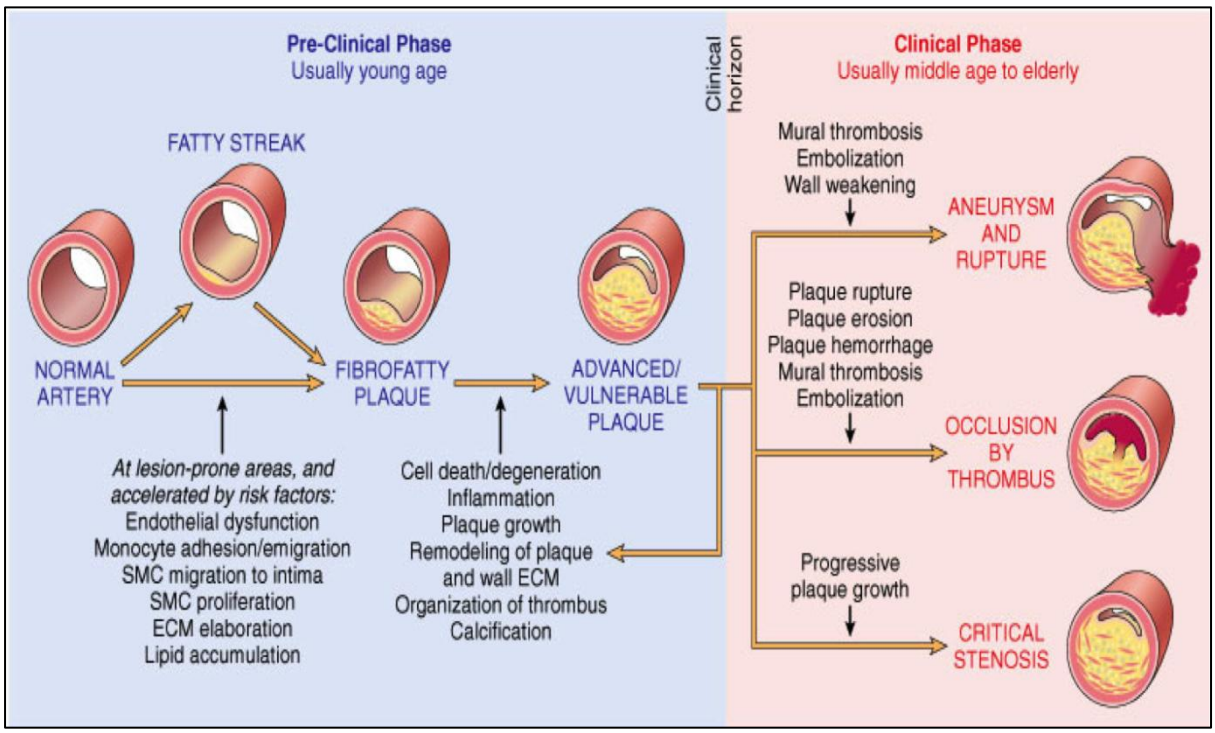
Early atherosclerosis

Subtle atherosclerosis

Advance atherosclerosis

Unstable coronary plaque [5]

Diagram no 8: natural history of atherosclerosis



VIRCHOW'S TRIAD IN THROMBOSIS

The concept of Virchow's triad plays an important role in coronary thrombosis too. It consists of

- Hypercoagulable state
- Blood flow stasis
- Vessel wall injury

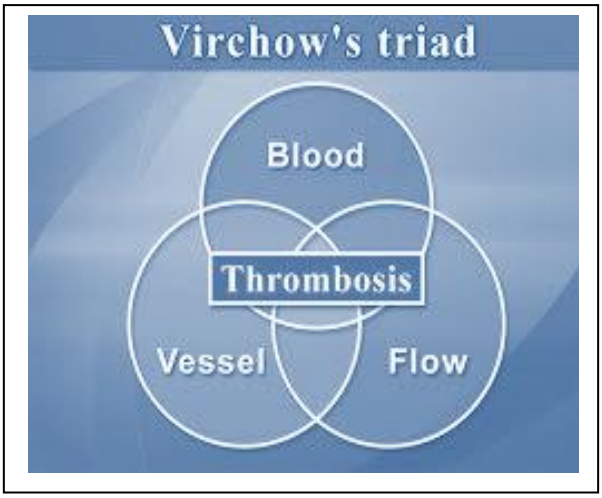


Diagram no 9: microscopic picture of a plaque rupture. Hematoxylin and eosin stain, showing occlusive thrombus overlying atheroma with rupture on shoulder (arrow) and communication of thrombus with atheroma core.

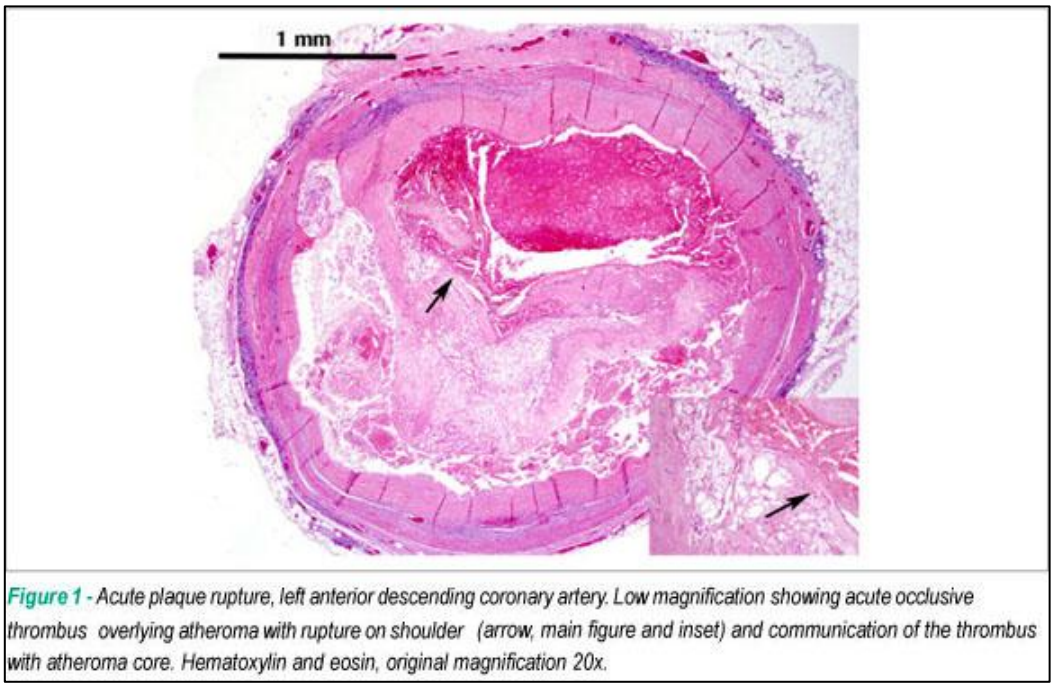


Figure 1 - Acute plaque rupture, left anterior descending coronary artery. Low magnification showing acute occlusive thrombus overlying atheroma with rupture on shoulder (arrow, main figure and inset) and communication of the thrombus with atheroma core. Hematoxylin and eosin, original magnification 20x.

NATURAL HISTORY OF MYOCARDIAL INFARCTION

The clinical picture of acute coronary syndrome may vary depending upon the fate of the ruptured atherosclerotic plaque (7)

- Complete occlusion results in trans mural (ST elevation) MI
- Partial occlusion results in unstable angina / non ST elevation MI
- Sometimes spontaneous resolution can occur

HAEMODYNAMIC ALTERATIONS

Myocardial infarction results in decreased contractility of heart and there by reduced stroke volume that stimulates the baroreceptors to increase the sympathetic outflow. This sympathetic surge increases the heart rate, increases after load, and decreases the diastolic filling, resulting in a vicious cycle. [6]

Classification of MYOCARDIAL INFARCTION

- Type 1: spontaneous myocardial infarction.

Atherosclerotic plaque rupture, ulceration, fissuring, erosion or dissection with resulting intra luminal thrombus in one or more coronary arteries is included in Type1.

- Type 2: myocardial infarction secondary to ischemic imbalance

Myocardial injury caused by conditions other than CAD, leading to imbalance between myocardial oxygen supply and demand ex: coronary endothelial dysfunction, coronary artery spasm, embolism, anemia, arrhythmias etc.

- Type 3: myocardial infarction resulting in death when biomarker values are available

Cardiac death with symptoms suggestive of ischemia and presumed new ischemic ECG changes before blood samples could be collected.

- Type 4a: myocardial infarction related to percutaneous coronary intervention

It is arbitrarily defined as elevation of cTn values >5 *99^{th} percentile URL in patients with normal baseline values (<99^{th} percentile URL) or a rise of cTn values >20% if the baseline values s\are elevated and are stable or falling. In addition to this 1) symptoms suggestive of myocardial ischemia 2)new ischemic ECG changes

3)angiographic loss of patency of a major coronary artery or a side vessel or a 4)imaging demonstration of new loss of viable myocardium or chest wall abnormality is required.

- Type 4b: myocardial infarction related to stent thrombosis

It is detected by coronary angiography or autopsy with a rise and/fall of cardiac biomarkers with at least one values above 99th percentile.

- Type 5: myocardial infarction related to coronary artery bypass grafting

It is defined by elevated cardiac biomarkers >10* 99th percentile URL. In addition- either 1) new pathological Q waves or new LBBB or 2) angiographically documented new graft or new native coronary artery occlusion or 3) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

CLINICAL FEATURES

Diaphoresis, nausea, and vomiting, cold peripheries, delirium, giddiness, abdominal discomfort, unexplained syncope attacks [8]

Symptoms

Crushing pain lasting usually for 15m inutes or more with or without

- Nausea , vomiting , abdominal blotting
- Pallor
- Sweating , fatigue
- Feeling of impending death
- Breathing difficulty

SIGNS:

- Rapid thready (low volume) pulse

- Elevated blood pressure due to pain
- Low blood pressure due to cardiogenic shock
- Elevated jugular venous pulse
- Peripheral and central cyanosis
- Febrile because of ongoing inflammation
- Tachypnoea in left ventricular dysfunction
- Tachycardia or bradycardia (inferior wall MI)
- Lung crepitations in pulmonary edema
- Muffled heart sounds especially S1
- Systolic murmur due to ventricular septal rupture or papillary muscle rupture

LABORATORY INVESTIGATIONS

- **CREATINE KINASE(CK) :**

Serum creatinine kinase peaks at approximately 24 hours. It starts to elevate within four to eight hours. However reperfusion either by fibrinolytic therapy or surgical recanalization can influence the enzyme values by influencing infarct size or time activity graph of the enzyme [13]

CK-MB iso-enzyme immunoassay is highly sensitive and specific for ischemic heart disease .A ratio of 2.5 between creatinine kinase MB and creatinine kinase is highly indicative for myocardial injury

- **Troponins**

Troponins are proteins that help in muscle contractions , they are present in both skeletal and cardiac muscles. However the main acid sequences in the cardiac and skeletal troponins are different. Therefore quantitative assays using antibodies against cardiac troponins (c TnT and c TnI) can be used for the diagnosis of MI

The levels of c Tnt & c TnI starts to rise by 3 hours of experiencing angina. The levels of c TnI and c TnT remain elevated for 7- 10 days and 10-14 days respectively.

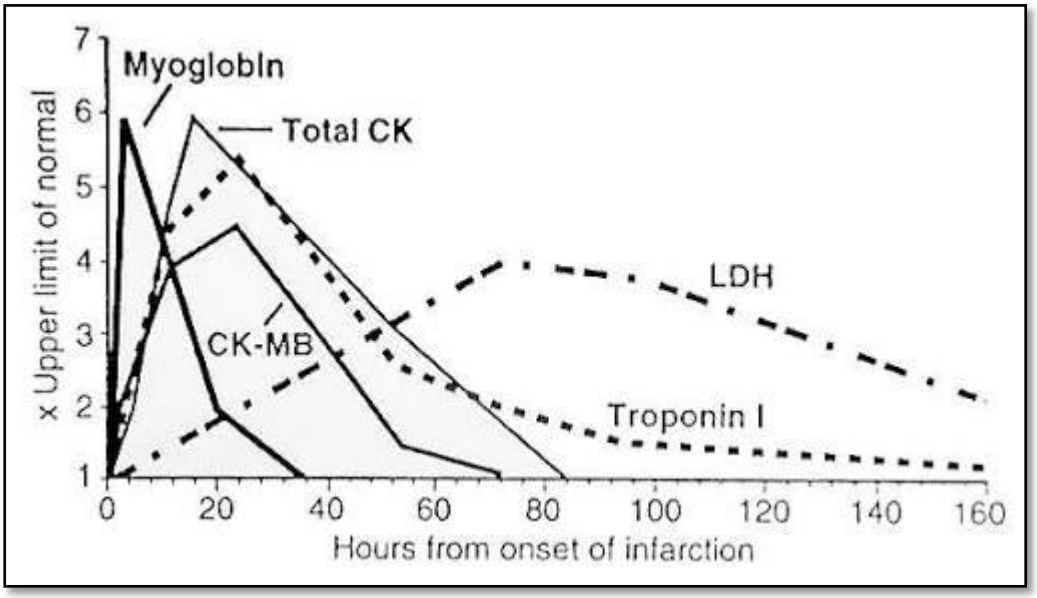
ADVANTAGES OF CARDIAC TROPONINS

- **Remain elevated for a long time ,thus helping in diagnosis in those presenting late**
- **As an indicator of reperfusion – if reperfusion is successful ,there is a sudden release of large amount of cardiac troponins**
- **Cardiac troponins can detect degrees of cardiac tissue necrosis which are below the range of creatinine kinase MB.**

Diagram no:10 Cardiac markers

Markers	Time of Initial Elevation	Time of Peak Elevation	Time to Return to Normal
CK-MB	4-8 h	12-24 h	72-96 h
Myoglobin	2-4 h	8-10 h	24 h
Troponin I	4-6 h	12 h	3-10 days

Diagram no :11 graphical representation of rise and fall of cardiac biomarkers



Emerging biomarkers

<u>Brain natriuretic peptide and N-terminal pro brain natriuretic peptide</u>

Like atrial natriuretic peptide, BNP is also secreted from the heart in response to stretch receptors within the muscle wall. Here BNP and Pro BNP have become well established biomarkers for left ventricular dysfunction.

However, they are also being investigated as prognostic indicators of long term mortality early after an acute coronary event. This was applicable to entire spectrum of acute coronary syndrome, including STEMI, NSTEMI and unstable angina. A trend

towards favorable outcomes was seen in a sub study of FRISC II trial following an early invasive strategy in patients with high values of NT-Pro BNP.

C-reactive protein

C-reactive protein (CRP) is an acute phase reactant protein secreted by the liver in response to acute inflammatory conditions. Recent studies have compared the values of CRP and the other established biomarkers in patients with ACS.CRP can be used by the clinician when risk stratification with additional prognostic information is desired.

Developing biomarkers

The present biomarkers have ailed physicians in diagnosis and prognostication of ACS but there are areas where they are found to be deficient

- Low sensitivity in the initial 4-6 hours after onset of chest pain. This issue is being addressed by ultrasensitive troponins now
- In the absence of myocardial necrosis, they are poor markers for ischemia
- They are affected by injury or inflammation of other body organs.

A number of novel biomarkers re in the development stage in recent years.

Myeloperoxidase

MPO is a degranulation product coming from white cells. It may be involved in the development of lipid laden soft plaque, protease cascade activation and nitric oxide consumption. Thus MPO is an indicator of inflammation and plaque instability ,but not ischemia.

In one study, MPO level was an independent prognostic factor for outcome at 6 months in terms of death and recurrent MI. even though various studies have shown a positive correlation between MPO levels and a worse outcome, more studies are required to establish the efficacy of MPO in detecting mild ACS and adverse prognosis.

Soluble CD40 ligand

It is a signaling protein that reflects both inflammatory and platelet interactions within the plaque.

Matrix metalloproteinases(MMP)

These are a class of endopeptidases that are_regulators of the extracellular matrix (ECM). MMP- 9 is localized in the shoulder of the plaque which is the thinnest portion of the plaque. *Blackenburg et all* examined the potential of MMP-9 in the risk stratification of people with ACS. Higher values were found to have a positive correlation with adverse myocardial events in the future.

Fatty acid binding proteins

They are the major vehicles for cytosolic transport of long chain unesterified fatty acids. Heart type fatty acid binding proteins (H-FABP) appears in the blood soon after MI (within 2-3hours) and returns to normal (within 12-24hours) in patients without renal impairment.

It has been suggested that FABP can be used in diagnosis of chest pain of uncertain origin and as a marker of ischemia when there is no myocardial necrosis. However lack of specificity, constraints its use.

Free fatty acids unbound to albumin

It has been evaluated for early identification of cardiac ischemia. Several studies have implicated the sensitivity of this marker on admission to emergency room and have shown that free fatty acids unbound too albumin evaluated earlier than the traditional biomarkers in 100% of the patients.

Ischemia modified albumin

Levels of Ischemia modified albumin (IMA) holds a strong negative predictive value for ischemia even prior to necrosis. This is based on the theory that metal binding sites on albumin are altered on exposure to ischemia. When combined with cardiac Troponin T and ECG, IMA is extremely useful to rule out ACS. However due to the lack of a gold standard to evaluate non necrotic ischemia, it has been difficult to assess IMA

Pregnancy associated plasma protein 3

This is a member of insulin like growth factor family of proteins and is considered to be elevated in neo-vascularisation. This PAPP-A protein may be used as a marker for impending plaque rupture.

Placental growth factor

It is a member of vascular endothelial growth factor (VEGF) and was shown to be elevated in early and late atherosclerotic plaque. Placental growth factor (PIGF) is mainly involved in the inflammatory process of atherosclerosis. However like PAPP-A , no standard assays are available to assess the efficacy of these markers.

MYOGLOBIN

The levels of myoglobin are elevated before CK-MB and cardiac troponins. However due to its lack of cardiac specificity, myoglobin values alone are not sufficient in making a diagnosis of MI

SERUM LIPIDS

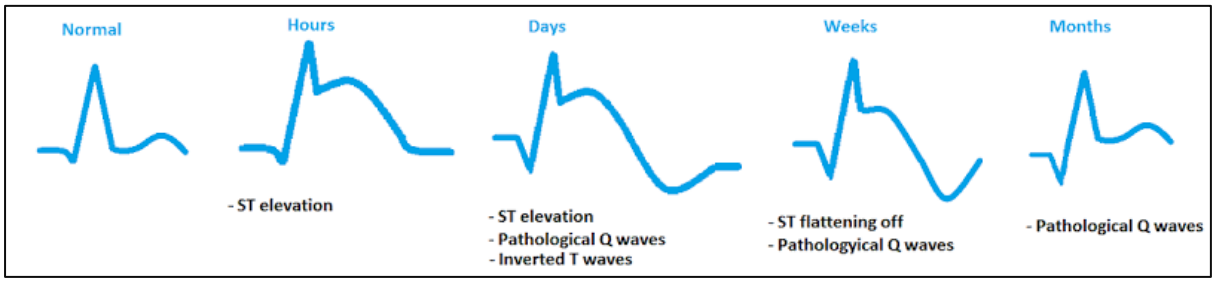
A lipid profile should be obtained in all patients who are admitted within 24- 48 hours of symptoms. This can help in risk assessment. However, their values start to fall after 24 -48 hours; the fall in HDL value is greater than the fall in cholesterol. So the ratio of HDL to cholesterol cannot be taken for risk assessment if measured late.

HEMATOLOGICAL FINDINGS

Leukocytosis occurs within two hours of developing symptoms. ESR starts to rise usually 1 or 2 days after the onset of symptoms and remain elevated for several weeks. It is unrelated to the prognosis and size of infarct

ELECTROCARDIOGRAM

Diagram no: 12 ECG changes in MI over a period of time



ECG is a basic diagnostic modality in the treatment of MI.

For the diagnosis of STEMI, an elevation of 1mm or more in ST segments of two or more contiguous leads is necessary. A reciprocal change (ST depression) in leads leading away from the site of infarct further confirms the diagnosis. [9]

ECG Changes

INFERIOR WALL MI– elevation in LEAD II and LEAD III with ST depression in lead I, avl or both.

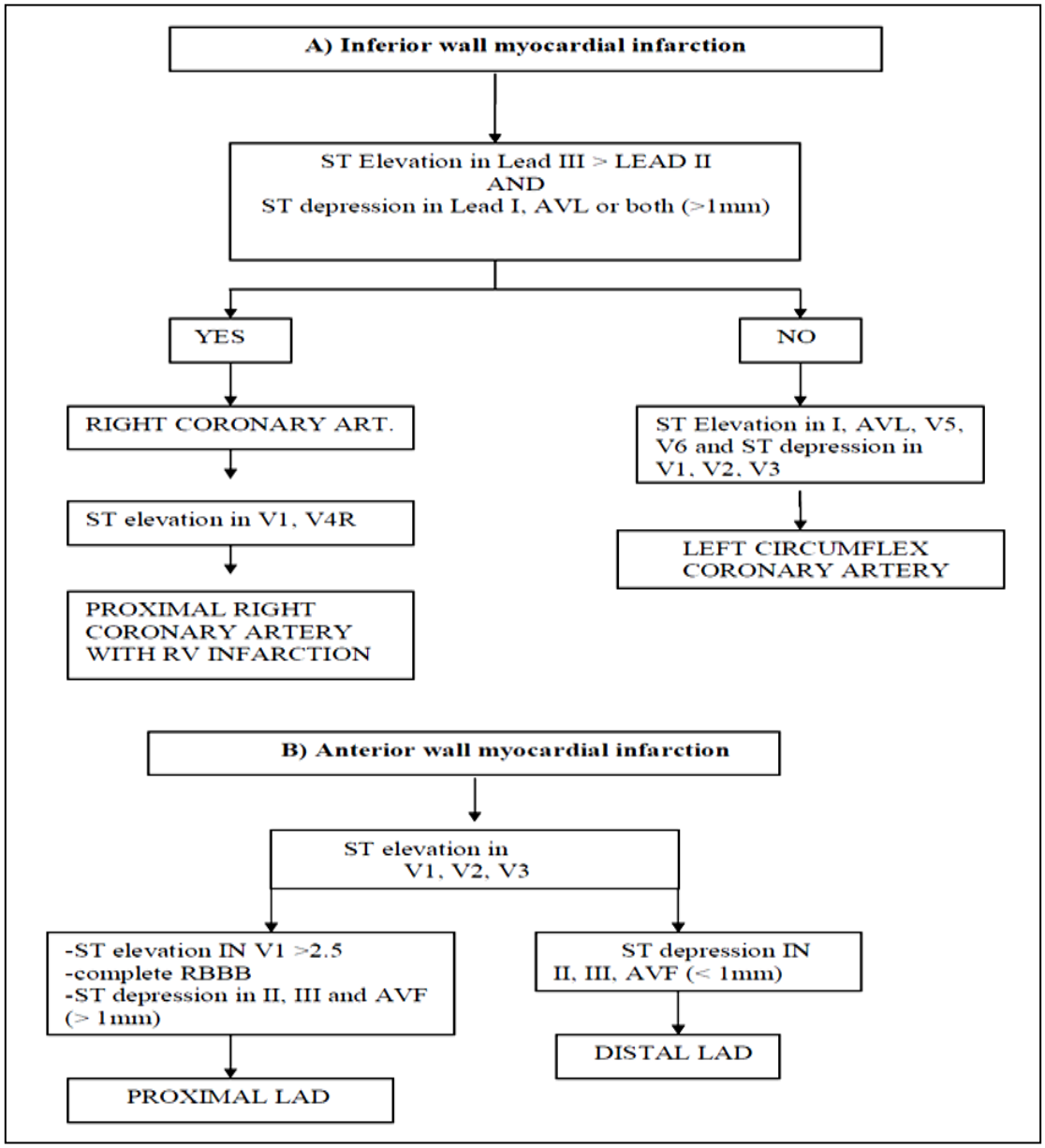
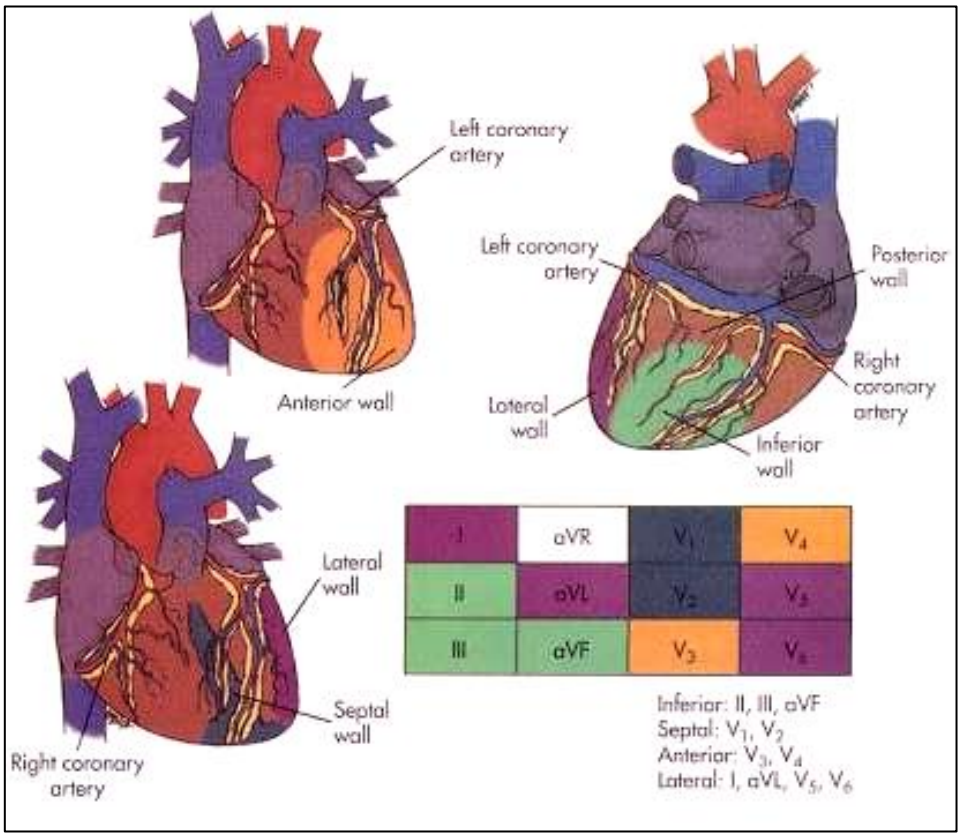


Diagram no: 13



Patients with NSTEMI can present with marked or minimal ST-segment depression or isolated T wave changes. This identifies patients at higher risk who subsequently benefit most from an aggressive management strategy [10].

Echocardiography

Abnormal wall motility is seen in all patents with MI and the degree of abnormality can be categorized with wall motion .An echocardiogram can help in assessment of risk , diagnosis in patients with symptoms suggestive of MI but normal to near normal ECG and the risk of developing congestive cardiac failure. [14]

Chest X ray

It helps in ruling out other diseases like aortic dissection which can mimic MI. It also helps to rule out pulmonary oedema which can develop as a result of systolic or diastolic dysfunction [12]

RADIONUCLIDE TECHNIQUE

It can detect infarction earlier than enzyme. This test is not generally used as it is expensive and cannot distinguish between a new infarct and an old scar.

POSITRON EMISSION TOMOGRAPHY

PET can easily differentiate stunned myocardium due to acute ischemia from an old scar .18 fluoro-deoxy glucose is used as a marter. PET uses positron emitting agents to demonstrate change in metabolism of the cardiac tissues.

CORONARY ARTERIOGRAPHY

It is indicated in

- Patients with persisting symptoms; unconfirmed diagnosis of MI
- chronic stable angina in spite of medical therapy
- angina patients who suffered cardiac arrest and survived
- patients with clinical or laboratory evidence of ventricular dysfunction [16]

Non invasive options include CT angiography and MR angiography. The high dose radiation of CT angiography and cardiac motion induced artifacts on MR angiography are the main limitations.

STRESS TESTING

One day prior to the test all medications including beta blockers are discontinued. A 12lead ECG is taken while the patient is made to exercise upto 85% of predicted heart rate. The endpoints are inability to exercise for more than 2min, a new ST depression of 2mm or more or development of heart failure, hypotension or arrhythmia.

Drugs like adenosine, dypiridamole and dobutamine can also be used instead of exercise.[15]

RISK STRATIFICATION AND PROGNOSIS:

Thrombolysis in MI Risk Score for Unstable Angina/NSTEMI

One Point score for each of the Following:	**Score**	**Risk of Adverse Event[*]**
Age ≥65 years	0/1	4.7%
Presence of =3 CV risk factors	2	8.3%
Recent (<24 h) severe angina	3	13.2%
Known coronary stenosis ≥50%	4	19.9%
ST-segment deviation on admission ECG ≥0.5 mm	5	26.2%

Thrombolysis in MI Score for STEMI

Various Risk Factors	**Points**	**Total Score**	**30-Day Mortality rate (%)**
Patient Age ≥75 years	3	0	0.8
Patient Age 65-74 years	2	1	1.6
Systolic Blood Pressure <100 mm Hg	3	2	2.2

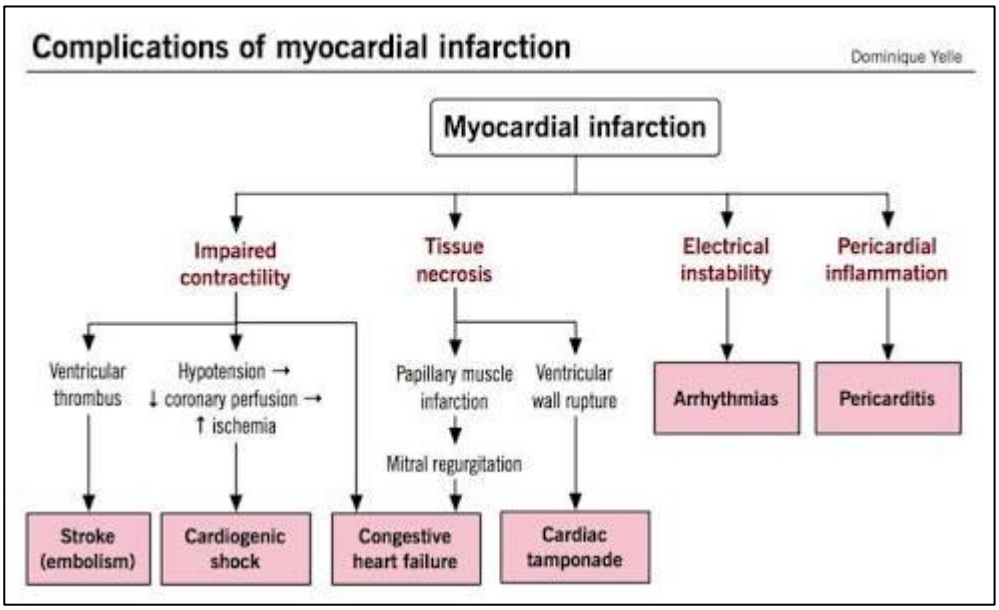
Pulse rate>100 beats/min	2	3	4.4
Killip class more than 1	2	4	7.3
Anterior MI or LBBB	1	5	12.4

Abbreviations: BP = blood pressure; HTN =hypertension; LBBB = left bundle branch block; MI = myocardial infarction

COMPLICATIONS OF MI

- Pericarditis
- Cardiac tamponade
- Arrhythmias
- Cardiogenic shock
- Congestive heart failure
- Stroke

Diagram no: 13 summary of complications of MI



MANAGEMENT

Basic steps include –

- Establishing an IV access and stabilising the patients
- Drug therapy include
  - Control of pain by sublingual nitrates, nitroglycerin or IV morphine

Nitroglycerin it can be given as a sublingual or intravenous dose. It helps in alleviating pain. It causes coronary vasodilatation and decrease in venous preload. However it should preferably not used in patients with inferior wall and right ventricular MI. it is contraindicated in patients with hypotension and those who have used PDE inhibitors within 24 hours

Intravenous Morphine of 2-4mg: it reduces pain, decreases myocardial oxygen demand, reduction in sympathetic tone and alleviates anxiety of the patient

- Minimize myocardial oxygen demand

Beta blockers reduce pain, reduce oxygen mismatch by reducing heart rate, decrease the chance of developing arrhythmias and even decrease infarct size .Oral beta blocker therapy should be started in all patients in the absence of contraindications. Metaprolol is used because of its short half life

Intravenous beta blockers are used in acute AC after excluding patients in heart failure, hypotension (< 90mm Hg), bradycardia (<60 bpm) or heart block

- Maintain myocardial perfusion

Aspirin

It is an irreversible cyclo-oxygenase inhibitor. All patients with MI should receive a loading dose of 300mg followed by 150 mg .it reduces mortality in patients with ACS

Clopidogrel

It is an anti-platelet drug. Combined with aspirin it reduces the incidence of re infarction, stroke and cardiovascular death

Heparin

It acts by activating anti thrombin III, inhibits thrombin and factor IX a. In patients with ACS 70IU/kg bolus IV followed by an infusion rate 12-15 IU/kg/Hour infusion rate is maintained. [17]

Reperfusion therapy

A 12 lead ECG is the primary tool in the diagnosis of acute coronary syndrome. The time from symptom onset to reperfusion is the most important determinant in the latter's success, whatever the modality of reperfusion may be.

In the absence of ST elevation, thrombolysis is ineffectual and can be harmful. If the patient presents within 3 hours of onset of symptom and there is no delay in invasive strategy, either strategy can be tried

Fibrinolysis

It is preferred when presentation is within 3 hours and PCI is not a preferable option due to difficulty in vascular access, delay in transportation and lack of catheterization facilities.

The agents used are streptokinase, alteplase etc

Contraindications and Cautions for Thrombolysis in STEMI [19]:

- *History of ICH*
- *Ischemic stroke within 3 months*
- *Suspected aortic dissection*
- *Active bleeding or bleeding diathesis (excluding menses)*
- *Intracranial neoplasm or vascular anomaly*
- *Head or facial injury in the last 3 months*

Relative contraindications:

- *severe hypertension (>180/110 mm hg)*
- *History of ischemic stroke >3 months*
- *Recent (within 2-4 wk) internal bleeding*
- *Pregnancy*
- *Active peptic ulcer*
- *Current use of anticoagulants INR > 2.0*
- *Major internal surgery within3 weeks*
- *Prior streptokinase exposure*
- *CPR for greater than 10 min*

Percutaneous coronary intervention

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It is the treatment modality of choice in patients with contraindications for thrombolysis. It is the preferred mode in patients in cardiogenic shock as it carries a lower risk for hemorrhagic complications [20]

PCI is chosen when

High risk from STEMI

- Cardiogenic shock

- Killip class ≥3

- Contraindications to fibrinolysis including increased risk of bleeding, ICH

-Late presentation

- Symptom onset was more than 3 hr ago

-Diagnosis of STEMI is in doubt.

FEMALE RELATED STATISTICS

HYPERTENSION:

Framingham data revealed that women with systolic blood pressure greater than 180 had an annual incidence of coronary events > 30% while in men it is >50%.

The probability of developing hypertension increase with advancing age, since women have a longer life expectancy than men, there are more elderly women with hypertension [23]

Diastolic blood pressure also is an important predictor of ACS as predicted by other clinical studies.

The pharmacotherapeutic and pharmacodynamic properties of ACE inhibitors and thiazide diuretics in relation to gender is under study. The common side effects of ACE inhibitors like cough are more common in females than males. [30] It is important to note that ACE inhibitors have teratogenic potential and are contraindicated in pregnant women especially in the first and second trimester. [29]

Thiazide diuretics have been proven to be beneficial in menopausal women due to its effect on bone mineral density. Studies have shown that it decreases the incidence of hip fractures by approximately one third. [24]

LIPIDS

An abnormal lipid profile is an important tool in the risk estimation of both men and women. The lipid profile of both sexes is different and also varies in their impact on cardiovascular events. [31]

The co-relation between low HDL and cardiovascular events is more significant in women than males compared to other lipoprotein components; HDL values are more predictive for females.

The pharmacologic control of hyperlipidemia after an episode of MI has been proven to be beneficial in both sexes. However, it is observed that the dosage of Hmg CoA inhibitors used in women is often inadequate and target levels are not reached. The recent cholesterol control guidelines recommend aggressive primary prevention of hypercholesterolemia in diabetic females.

DIABETES

Even though mortality rates after ACS have generally been declining in the past decade, mortality rates in diabetic women with ACS have increased by 23 percent while it has decreased by 27 percent in non diabetic females. It is quite clear that diabetes predisposes to serious complications after MI and mortality. (21)

Higher in-hospital mortality and congestive heart failure is seen among women than men. The presence of hypertension in diabetic patients both male and female further increases the mortality rates. (34)

Diabetes confers substantially increased relative risk of first incident and admission for MI in women above 65 years of age compared with age to sex matched controls younger than age 65 years over 20 years follow up in Copenhagen City heart Study (35).

Altered lipid profile is highly common in diabetic patients. A low level of HDL correlating with increased incidence of coronary events is more common in diabetic females. Other lipoprotein components are also elevated. Use of HMG Co A Inhibitors for hyperlipidemia in diabetic patients has resulted in fewer incidence of coronary vascular event.

Gestational diabetes and obesity predisposes to the development of diabetes. The possibility of developing diabetes can be attenuated by even a moderate increase in physical exertion and weight reduction. (37)

INSULIN RESISTANCE SYNDROMES

The insulin resistance condition comprises a spectrum of disorders, with hyperglycemia representing one of the most readily diagnosed features. The *metabolic syndrome*, the *insulin resistance syndrome*, or *syndrome X* are terms used to describe a constellation of metabolic derangements that includes insulin resistance, hypertension, dyslipidemia (decreased HDL and elevated triglycerides), central or visceral obesity, type 2 diabetes or IGT/IFG, and accelerated cardiovascular disease[41].

Mutations in the insulin receptor that interfere with binding or signal transduction are a rare cause of insulin resistance. Acanthosis nigricans and signs of hyperandrogenism (hirsutism, acne, and oligomenorrhea in women) are also common physical features. Two distinct syndromes of severe insulin resistance have been described in adults: (1) type A, which affects young women and is characterized by severe hyperinsulinemia, obesity, and features of hyperandrogenism; and (2) type B, which affects middle-aged women and is characterized by severe hyperinsulinemia, features of hyperandrogenism, and autoimmune disorders. Individuals with the type A insulin resistance syndrome have an undefined defect in the insulin-signaling pathway; individuals with the type B insulin resistance syndrome have autoantibodies directed at the insulin receptor.

These receptor autoantibodies may block insulin binding or may stimulate the insulin receptor, leading to intermittent hypoglycemia.

Polycystic ovary syndrome (PCOS) is a common disorder that affects premenopausal women and is characterized by chronic anovulation and hyperandrogenism. Insulin resistance is seen in a significant subset of women with PCOS, and the disorder substantially increases the risk for type 2 DM, independent of the effects of obesity.

HYPERGLYCEMIA IN MYOCARDIAL INFARCTION:

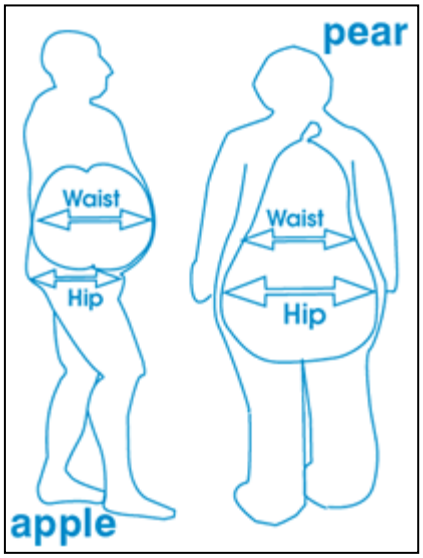
The mortality and morbidity of the diabetic patient sustaining a myocardial infarction is poor compared with that of non-diabetic patients. Studies have shown that hyperglycemia induces adverse prognosis even in non diabetic patients. Claure Bennard observed and explained acute hyperglycemic response to stress more than a century ago. “Diabetes of injury” concept evolved as glucose has been identified as metabolic mirror of the severity and outcome of critical illness.

OBESITY

The prevalence of obesity is on the rise in our country. This can be attributed to the hazardous change in diet and lifestyle of the people.

Obesity is one of the most important modifiable risk factor in the development of ACS. Along with this, it is related to the other cardiac risk factors like diabetes, hyperlipidemia, and metabolic syndrome. [39] An increased waist circumference has comparatively more impact than a raised BMI. [41] The type of weight distribution also influences the risk, i.e. apple shaped abdomen is more dangerous than pear shaped abdomen.

Diagram no: 13



Even though many revolutionary drugs have come into the market for the treatment of obesity, the adverse effects often outweigh the benefits.

PHYSICAL ACTIVITY AND EXERCISE:

Exercise and physical activity is important for both primary and secondary prevention of vascular events. [47] Studies have proven that women who walk for 1 hour each week have lesser incidence of cardiac events than women who d not walk regularly. However, studies on the influence of exercise usually focus on leisure activities like yoga aerobic etc and exclude household work and child care. This can lead to underestimating the actual energy expenditure of women.

MENOPAUSE AND HORMONAL THERAPY

The effect of menopause and hormonal therapy on cardiovascular events is still under study. Coronary artery disease is the leading cause of death in post menopausal women. Gynaecological surveys have shown an increased risk of developing ACS in women with early menopause due to reduced hormone exposure

The Women's Health Initiative (WHI) study investigated health risks and benefits of hormonal therapy in healthy menopausal women aged 50 – 79 years old. [49] In the study

- Continuous combined HRT (CEE 0.625 mg + MPA 2.5 mg) was given to women with intact uterus. The study which was originally supposed to run for 8.5 years was stopped after 5.2years because the harm (CHD , stroke, pulmonary embolism) outweighed benefit
- Oestrogen alone was given to women with previous hysterectomy. This study was also discontinued due to no heart disease benefit and increased stroke risk.

The HERS study (Heart and oestrogen progestin replacement study) conducted earlier was the first secondary prevention clinical trial which demonstrated that hormone replacement therapy showed no benefit in the prevention of coronary heart disease. These postmenopausal women had an incidence of coronary artery disease (myocardial infarction, coronary artery bypass grafting, percutaneous transluminal coronary angioplasty for occlusion >50 percent, or angiography with more than one major coronary artery)

Based on the above studies, national guidelines have stressed the importance of other modalities of prevention over hormone replacement. In counselling post menopausal women, cardiovascular prevention should be given importance.

PSYCHOSOCIAL FACTORS

Emotional stress plays an important role in predisposing an individual to CAD as well as in increasing morbidity and mortality after a cardiovascular event. The Nurses' health study has revealed that sudden cardiac death is associated with phobic anxiety which is more common in women. Acute and reversible cardiomyopathy following stress has also been proven.[50] Women with lower socioeconomic status has a higher probability of developing CHD and higher mortality which is often due to the delay in treatment and improper understanding of the severity of the disease.

Women are more prone to develop depression than men. Interestingly, news of cancer is less likely to cause depressive feelings than a myocardial infarction. Depression is

more likely to develop in young women with co-morbidities and low socio economic status, evidenced by **Prospective Registry**

Evaluation Outcomes after Myocardial Infarction: Events and Recovery (PREMIER) study. Women diagnosed with depression are generally undertreated.

TOBACCO

Irrespective of age and gender, tobacco exposure is an important risk factor in the development of CAD. It is important to note that incidence of CHD increases proportionally with the amount and duration of smoking in a dose related fashion.

In women, smoking leads to earlier onset of menopause and MI. the danger of smoking becomes clear when it is contemplated that women are the usual candidates for asymptomatic MI.

Potential weight gain on cessation of smoking is an important discouraging factor for women. However many interventions have been developed which can help with this problem. This includes proper counselling, physical activity and pharmacotherapy. Increase in physical activity itself helps in quitting smoking.

Pharmacological approach includes nicotine replacement and bupropion. Nicotine replacement increases the chance for success in smoking cessation. bupropion has an antidepressant action; it helps in cessation of smoking and has been found to be beneficial in minimizing weight gain. It should be avoided in patients with history suggestive of anorexia/ bulimia, heavy alcohol consumption, seizures and head trauma

RACIAL AND ETHNIC FACTORS

The importance of racial, social and ethnic factors in the development of cardiovascular diseases has recently come into the limelight. According to the 1986 National Mortality Feedback Survey, the 1985 National Health Interview Survey, and the U.S .Bureau of the Census Black women aged less than 55 years have a higher cardiovascular mortality than young American women and young men.

The data on Asian women and those living in India is insufficient, however the recent years have shown an increase in the number of young women and men presenting with MI

SIGNIFICANT COMORBIDITIES

Higher risk of coronary artery diseases is seen in patients with inflammation of coronary vasculature.

The potential of SLE and rheumatoid arthritis to act as an etiological factor in CAD has been highlighted

SLE (systemic lupus erythrematosus):

There is five-fold increased risk for developing CAD in patients with SLE.

Cardiac ultrasound done on 197 SLE patients and matched controls showed

That 37.1 percent of SLE patient had cardiac plaques. Those with cardiac plaques had a longer duration of SLE, generally older than 40, with inadequate treatment with anti inflammatory drugs.

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Rheumatoid arthritis:

Rheumatoid arthritis increases the mortality and morbidity in cardiovascular events. 98 women with rheumatoid arthritis were assessed with cardiac ultrasound for the evidence of atherosclerosis. 44 percent of these women showed the presence of plaques as compared to 15 percent age matched hypertensive women. Anti-inflammatory drugs like methotrexate have been shown to reduce cardiovascular mortality in rheumatoid arthritis.

INTERVENTIONS FOR CORONARY ARTERY DISEASE:

According to the recent statistics, 33 percent of patients treated with PCI are women. Complications both short and long term are more common in females than males. this might be related to the higher incidence of co morbidities, longer duration of symptoms and older age in females. Vascular complications are also more prevalent in females. [51] The advent of smaller coronary catheters has slightly decreased the gender specific variations in complications.

Statistically, men underwent CABG more commonly than women. [51] Experiments have shown that women with off-pump CABG were having older age and smaller surface area. Despite controlling these factors and other co morbidities women showed higher incidence of wound infections and longer in-patient stay but decreased mortality.

SUDDEN DEATH

Phobic anxiety which is more common in women than men increases the risk of fatal complications of CHD and sudden death. The Nurses' health study found that phobic

anxiety correlated with smoking, diabetes, hypertension, hyperlipidemia and BMI>30kg/m2.[52]

Arrhythmias

Women respond to the need in increasing cardiac output during physical exercise by increasing heart rate.[53] Women have a higher heart rate than men and studies have shown that after excluding potential confounders like diabetes, hypertension, previous CHD , anaemia and smoking, women with HR >60 bpm have higher cardiovascular mortality rates

The type of arrhythmia predominant in males and females varies. Men are prone to develop atrial and ventricular fibrillation related with WPW syndrome, while women are more prone to develop atrio-ventricular node re-entrant tachycardia and orthodromic supraventricular tachycardia.

Drug-induced torsade de pointes occurs more commonly in women than men and the former have a higher risk of dying of atrial fibrillation.

In an important randomized control study done in women with atrial fibrillation, rate control has shown to be more beneficial than rhythm control despite prior electrical cardioversion.

DIAGNOSIS OF CAD IN WOMEN

Further evaluation is necessary after establishing a diagnosis a coronary artery disease. The earliest known non invasive technique of exercise stress testing shows low specificity and sensitivity for women.

Severity of coronary heart disease can be assessed in women with stress imaging techniques. Breast tissue can cause soft tissue attenuation in nuclear stress perfusion using thallium, so technetium is used.

Cardiac catheterization is less likely to show CAD in women so stress imaging techniques are usually preferred.

UNSTABLE ANGINA AND NON-ST SEGMENT ELEVATION MI

Definition

Stable angina is defined as chest or arm discomfort precipitated by emotional stress or exercise it is relieved within 15minuts by rest or sublingual nitroglycerin. Unstable angina has at least one of the three features.

- Occurring at rest and lasting >20 minutes
- Crescendo angina

Classification

The branwald clinical classification gives three groups

- A – primary angina
- B – secondary angina
- C-post mi unstable angina
- Class1angina-new onset of severe angina or accelerated angina no rest pain
- Class2-angina at rest with in the past month but not within 48 hours
- Class3 –angina at rest within 48 hours

Classification according to aetiology

- Plaque rupture and erosion

Rupture usually takes place at the shoulder region of the plaque because this area is infiltrated with inflammatory cells and subjected to high shear forces. High risk

plaques have thin fibrous cap and large lipid pool which increases the chance for rupture. In contrast fibrosis and calcification attenuates the risk for rupture. [6]

Erosion occurs centrally rather than at the shoulder. It appears to be more common among women who smoke whereas these plaque rupture occurs in hyperlipidemic men.

- Inflammation

It plays the most important role in plaque disruption. The monocytes, leucocytes and the endothelial cells in the plaque express adherent molecules which lead to accumulation of macrophages and T-lymphocytes. There is cytokines and chemokines as well as which degrades collagen that provides strength of the fibrous cap. [7]

- Infection

CMV, helicobacter pylori and Chlamydia pneumonia have been identified within atherosclerotic plaques. Also antibodies against Chlamydia heat shock proteins cross reacts with heat shock proteins produced by endothelium. However antibiotics regimens against Chlamydia have not shown any conclusive benefit.

- Platelet aggression

Platelets play a key role in the formation of unstable lesion from a stable atherosclerotic plaque there are three major steps in this process adhesion, activation, aggregation. [4] When a atherosclerotic plaque disrupts the collagen and tissue factor is

exposed to circulating blood and this leads to platelet adhesion via the platelet Gp1b receptor following its interaction with von willebrand factor and gpv1 binding to collagen.

The next step is platelet activation which consists of change in shape of the platelets, realise of platelet contents including thromboxine A2 and activation of gp2b/3a on their surface the next step is platelet aggregation in which cross linking of the platelet by fibrinogen occurs.

- Clotting cascade

After plaque rupture haemostasis is attained by the release of tissue factor. Thrombin is generated by the activation of factor X to Xa, this thrombin will convert fibrinogen in to fibrin activating factor XIII which stabilizes the fibrin coat leading to further propagation of the thrombosis. [(4)]

- Thrombosis

It has a great role play in the pathophysiology of UA/NSTEMI

- Coronary vasoconstriction

In contrast to the coronary artery segments of culprit lesion of patients with stable angina, culprit lesion in UA/NSTEMI have an increased response to vasoconstrictor stimuli. In prinzmetals variant angina focal spasm of a segment of epicardial coronary arteries are seen. The role of coronary vasoconstriction causing micro circulatory

angina resulting from constriction of small intramural coronary resistant vessel is evident

Secondary unstable angina

It occurs due to imbalance between oxygen supply and demand which is precipitated by condition like fever thyrotoxicosis , hypertension and aortic stenosis.

- Progressive mechanical obstruction

Unstable angina/NSTEMI can be caused by progressive luminal narrowing of the coronary arteries. This condition is usually seen in restenosis following PCI. However this can also occur without PCI and it is related to rapid cellular proliferation

Diagnostic tools

Other than history and clinical examination the following modalities are used.

- Electrocardiogram

It is the most important diagnostic tool in the evaluation of a patient with suspected NSTEMI ACS. [(2)] ECG helps in the diagnosis and also in the risk stratification based on pattern and magnitude of abnormalities. In UA, ST depression is transient and ST elevation and/or T wave inversion occurs in 30-60% of patients depending on the severity. Several studies have shown that new ST segment deviation even if only 0.05 mv is specific and an important measure of ischemia and prognosis. ST depression of

1mm is associated with 11% mortality and ST depression of >2 mm is indicative of 6 fold increased mortality

- Continuous ECG monitoring

About two thirds of all ischemic episodes, especially in the initial hours are clinically silent and thus not likely to be seen in a conventional ECG. Continuous computerised 12 leads ST segment monitoring is an important diagnostic tool

- Exercise or other stress test

In a patient with typical chest pain no stress test should be done in acute phase. However in a patient with inconclusive ECG, stress test has a better predictive value. A negative exercise testing has a high negative predictive value.

- Echocardiogram

It can detect transient localised abnormal wall motility during ischemia, it also helps in exclusion of aortic stenosis, aortic dissection, embolism or HOCM. Echo cardiography can also assess the LV systolic function which is an important prognostic marker in patients with IHD.

- Biochemical markers

  The elevation of biomarkers like CK-MB, troponin T and I identifies patients with NSTEMI. Patients with negative cardiac biomarkers with symptoms

highly suggestive of ACS should have biomarkers re-measured in the time frame 8 to 12 hours after the symptom onset.

- Imaging of coronary anatomy

The gold standard is invasive coronary angiography if revascularisation is considered it is absolutely essential to have angiographic imaging of coronary blood vessel. [(2)]CT angiography cannot be recommended as the coronary imaging modality of choice at present

- Other laboratory tests

Chest x ray is performed in the admission to rule out pulmonary congestion. Serum cholesterol value should be measured at the time of presentation they begin to fall by 30-40% within 24 hours of ACS.

The Newer anti-platelet drugs

- Prasugrel

It provides more profound and faster antiplatelet action than clopidogrel. However there was increased incidence of bleeding. Excess bleeding with prasugrel was not seen in diabetic patients since their platelets are known to be more hyper active

- Ticagrelor/azd6140

It is a reversible inhibitor of p2y12 receptor with half life of 12 hours. As compared to clopidogrel it reduces vascular death, non fatal MI or non fatal stroke. The use of this agent as associated with dyspnoea and ventricular pauses

- Cangrelor

It is a reversible potent short acting intravenous p2y12 receptor inhibitor with rapid onset of action. In the most recent CHAMPION PHOENIX trial cangrelor reduced the rate of ischemic events including stent thrombosis during PCI. It was not associated with increased bleeding complications.

- Thrombin receptor antagonists

It blocks platelet protease activated receptor. Ongoing TRA programs have shown a major decrease in cardiovascular events without any increase in bleeding.

MATERIALS AND METHODS

This study was conducted at Thanjavur medical college during the time period December 2014 to may 2015

- **It is a cross sectional study**

Source of data:

Consecutive cases of female patients admitted in the medicine casualty of Thanjavur medical college & hospital, who satisfied the inclusion criteria.

Sample size: 50

Inclusion criteria:

ACS in female patients diagnosed by

- Symptoms s/o ACS with definite ECG changes
- Or raised cardiac markers

Exclusion criteria:

- Chest pain with normal ECG and normal cardiac markers.

- 50 female patients who presented with symptoms s/o ACS with ECG changes and/or raised cardiac markers were studied with detailed history with special regard to history risk analysis.

Investigations:

CBC, RBS, RFT, fasting lipid profile, ECG, Troponin T and echocardiogram were done for these patients.

- Hypertension was documented either by history of hypertension or by a measurement value of > 140/90 mm Hg.
- Diabetes was documented either by history or a RBS value > 200 Mg/dl with importance given to the duration of diabetes and treatment
- Clinical symptoms at the time of presentation like chest pain, epigastric pain, palpitation and other symptoms were taken into account and patient stratification was done accordingly.
- Physical signs like hypotension, raised JVP, s3, s4 and rales were considered and used for classifying patients.

Patient stratification:

- According to age, patients were classified into 4 groups : <45, 45-60, 60- 75 and >75years.
- With regard to diabetes, patients were classified into non diabetic, diabetic onset <10 years and diabetic onset >10 years.
- Patients were also classified into hypertensive and non hypertensive.
- Lifestyle factors were also given importance. Based on occupation, these patients were classified into 3 activity levels as sedentary, moderate and heavy workers

ACTIVITY		
sedentary	moderate	heavy
Teachers, executives, tailors, pharmacists	Nurse,maids,cooli,basket makers,weaver,agricultural laborer ,beedi maker	Mineworker, wood cutter

- Lifestyle modifications among the 50 patients were also assessed and they were classified as regular exercise, irregular exercise, dietary planning and no lifestyle modifications
- Killip score is calculated based on classification according to the status of cardiac pump function, estimated clinically.

Class I – No signs of pulmonary congestion or shock

Class II – Moderate heart failure as evident by rales at the lung bases, S3 Gallop, tachypnoea

Class III- Severe heart failure (pulmonary oedema)

Class IV- Cardiogenic shock with systolic BP < 90 mmHg, peripheral Vasoconstriction, cyanosis, oliguria and confusion

- Patients were classified into STEMI, NSTEMI and unstable angina with the help of ECG and cardiac markers and the type of MI was determined according to surface ECG.

Description	ECG Leads With Changes	Artery Occluded
Inferior	II, III and aVF	RCA
Anteroapical	V3 and V4	Distal LAD
Anteroseptal	V1 and V2	LAD
Anterolateral	I, aVL, V5 and V6	Circumflex Artery
Extensive Anterior	I, aVL and V2-V6	Proximal LCA
True Posterior	Tall R in V1	RCA

- Fasting lipid profile was done for these patients and HDL was classified into < or equal to 20, 20 -30, 30 – 40, 40- 50 and >50 mg/dl. LDL values were classified into <130, 130- 160, >160 mg/dl.

- BMI was calculated by dividing weight in kg by height in metre square and it was classified into underweight, normal, over weight and obesity

  Class I, II AND III.

	BMI ($kg.m^{-2}$)	Health Risk
Normal	18.5 — 24.9	Normal
Overweight	25 — 29.9	Increased
Class I obesity	30 — 34.9	Moderate
Class II obesity	35 — 39.9	Severe
Class III obesity	> 40	Extremely high

- Statistical methods :

1. Diagrammatic (bar / pie chart) representation
2. Prevalence of risk factors and comparisons
3. Mean and standard deviation

- **Study end point**

The primary aim of the study is identifying the prevalent risk factors in the study population

LIMITATIONS

- It is hospital based one step study.
- Limited numbers of cases were studied.
- Patients were not followed up for future complications after discharge from the hospital.
- Limited number of risk factors were include in the study
- Physical exercise did into take into account energy expenditure in household activities and child rearing

- Important risk factors like SLE, RA, and thyroid disorders were not tested, only history was taken into account.
- Being a cross sectional study p value and risk ratio cannot be calculated.

However, the study can serve as a base for understanding the prevalent risk factors in women with CAD and the prime importance of implementing primordial prevention in the community.

OBSERVATIONS

CLINICAL PRESENTATIONS

Table no: 1

SYMPTOMS	NO. OF PATIENTS (TOTAL n= 50)
CHEST PAIN	39
RADIATION	12
BREATHLESSNESS	20
NO CHEST PAIN	11
EPIGASTRIC PAIN	10
NAUSEA	9
VOMITING	10
PALPITATIONS	10

OUT OF 50 PATIENTS ADMITTED WITH ACS, 78% of the patients presented with chest pain. Only 30.7% of those presenting with chest pain gave a typical history of squeezing or compressing chest pain with characteristic radiation.

22 % of the study group did not complain of chest pain.

Table no 2: age wise distribution of patients without chest pain

AGE(in yrs)	NO OF PATIENTS (n=11)
<45	0
45-60	3
60-75	4
>75	4

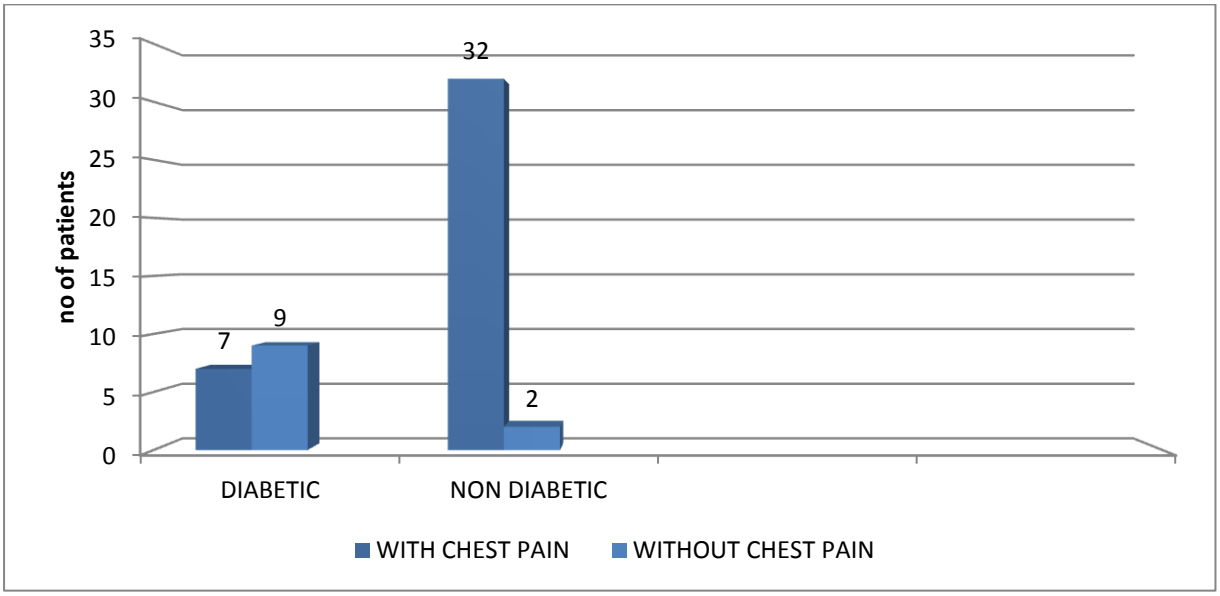
Out of the 11 patients without chest pain, 72.7% were above the age of 60 years

Table no 3: prevalence of diabetes in patients without chest pain

NO OF DIABETICS WITHOUT CHEST PAIN	9
NO OF NON DIABETICS WITHOUT CHEST PAIN	2
TOTAL(n =11)	11

Out of the 11patients, 82% were diabetics

Figure no: 1



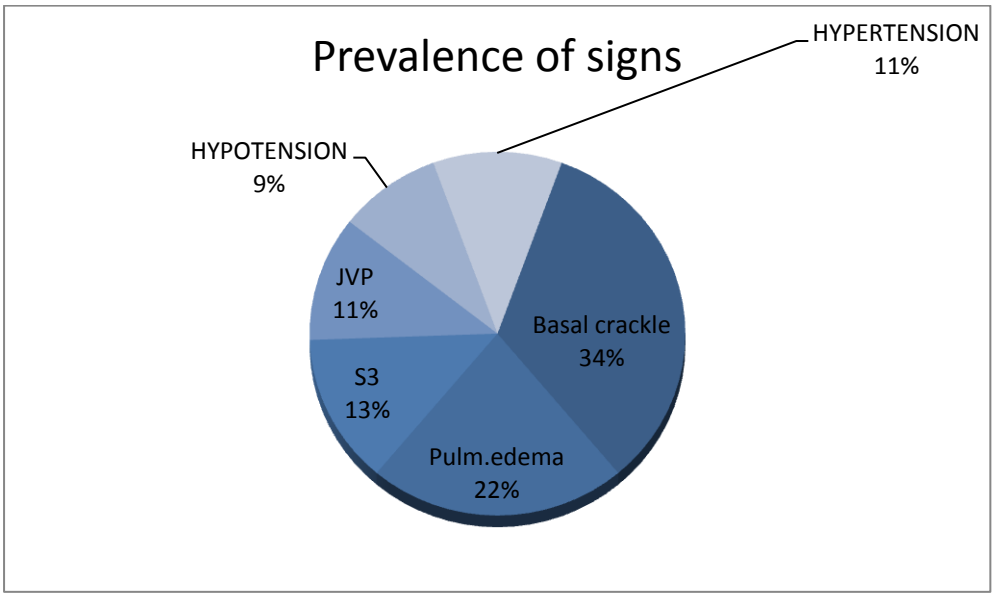
SIGNS

Table no 4: prevalence of signs

SIGNS	No of Patients (TOTAL=50)
CREPS/ BASAL CRACKLES	15
S3	6
S4	0
JVP	5
HYPOTENSION	4
HYPERTENSION	5
PUL.EDEMA/EXTENSIVE CRACKLES	11

The most common sign presented by this group of 50 patients was basal crackles/ crepitations i.e. 30%

Figure no 2



HISTORY

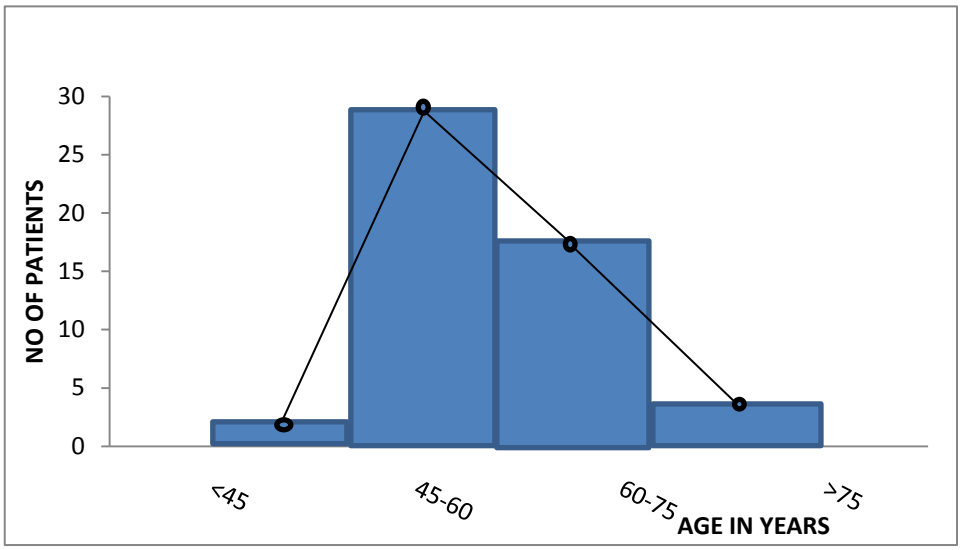
Age wise distribution of patients with ACS

Table no 5

Age classification	No of patients (total=50)
<45yrs	2 (4%)
45-60yrs	**27 (54%)**
60-75 yrs	17 (34%)
>75yr	4 (8%)

Out of 50 patients, 54% belonged to the age group of 45-60 years.

Figure no 3 Frequency polygon of age wise distribution of ACS in females



HISTORY OF DIABETES

Table no 6

Non diabetic	34(68%)
No of diabetics (>10years)	14(28%)
No of diabetics (<10years)	2 (4%)
Total	**16(32%)**

In our cross sectional study 32% of the population was found to have diabetes.

HYPERTENSION

Table no 7

No of normotensives	25
No of hypertensives	**25(50%)**
Total (n = 50)	

50% of the patients in the study population were diagnosed as hypertensives.

ROLE OF HORMONES

- Out of 50 female ACS patients, only 6 were in the premenopausal age.

44 of the patients had attained menopause.

I.e.**, 88 % of the study group had attained menopause.**

- Oral contraceptive pills

Out of 50 female patients, 8 **(16%)** gave a positive history of use of oral contraceptive pills on a regular basis.

LIFESTYLE RISKS

- Smoking and alcoholism

Only 2 among the 50 female patients gave a positive history of smoking, **7 gave a history of tobacco chewing** and 1 out of 50 gave a history of alcohol intake.

I.e., 18% (2 +7) gave a history of tobacco use

- occupation

Classification of activities according to occupation

Table no 8

Activity	No of patients (n=50)
sedentary	25(50%)
moderate	22 (44%)
heavy	3(6%)

50 % of the female patients in our study group led a sedentary lifestyle.

Classification based on lifestyle modifications

Table no 9

Lifestyle	No of patients (n=50)
Dietary planning	2(4 %)
Regular exercise	3(6%)
Irregular exercise	4 (8%)
No lifestyle modifications	**41 (82%)**

41 of the 50 female patients did not practice regular exercise nor any dietary planning

PAST HISTORY

Table no 10

HISTORY OF TIA/STROKE	4(8%)
HISTORY OF IHD	7(14%)
HISTORY OF RHD/SLE	NIL
HISTORY OF THYROID DISORDERS	3(6%)

In our study group of 50 patients, 7 gave a history of previous episode of IHD and 4 gave a history of TIA/stroke

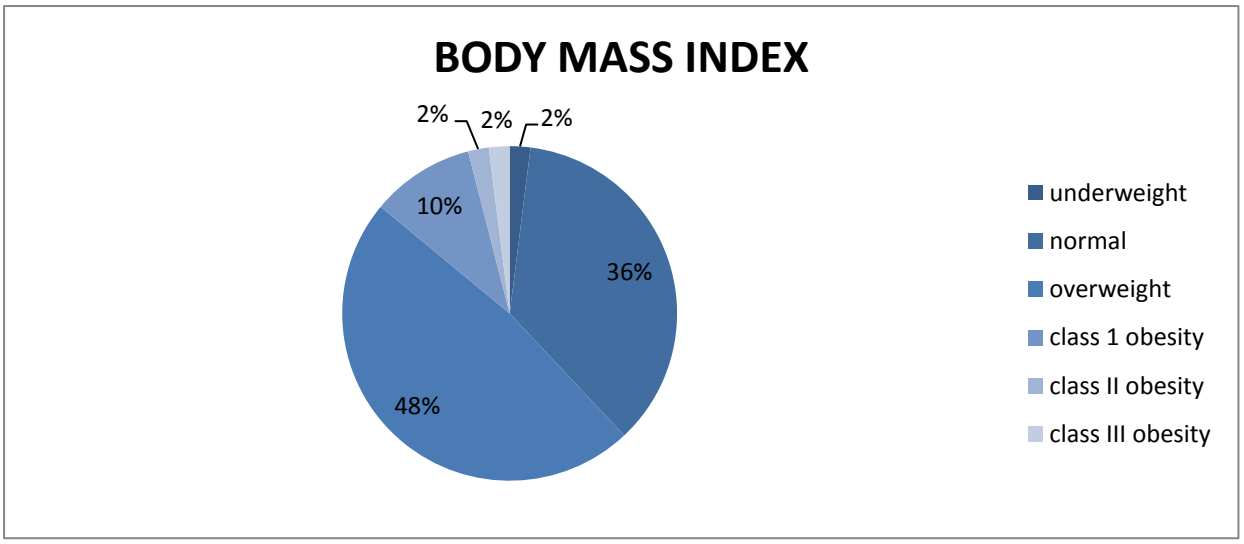
CLASSIFICATION BASED ON BMI

Table no 10

BODY MASS INDEX	NO OF PATIENTS (n=50)
<18.5	1
18.5-24.9	18 (36%)
25-29.9	24(48%)
30- 34.9	5(10%)
35-39.9	1(2%)
>40	1(2%)

Out of 50 patients, 48 % were overweight and 10% belonged to class 1 obesity.62% of the female patients with ACS has deranged BMI.

Figure no 6



FASTING LIPID PROFILE

Table no 11: high density lipid profile

HDL (mg/dl)	No of patients (n=50)
<20	1(2%)
20-30	24(48%)
30-40	17(34%)
40-50	7(14%)
>50	1(2%)

In the study, 50 % of female patient had a HDL value below 30.

Table no 12: low density lipid profile

LDL(mg/dl)	No of patients(n=50)
<130	19(38%)
130-160	11(22%)
>160	20(40%)

In the study, 40% of the study group had a LDL value above 160mg/dl.

CLASSIFICATION OF ACS PATIENTS BASED ON ST ELEVATION IN ECG

Table no 13: distribution of STEMI in the study group

ST elevation (STEMI)	30(60%)
No ST Elevation	20(40%)
TOTAL NO OF PATIENTS	50

30 out of the 50 female patients in the study group were diagnosed to have STEMI , while the remaining 20 had NSTEMI/UA.

Table no 14: classification of STEMI according to area affected

ECG finding(area affected)	No of STEMI patients n= 30
IWMI	12(40%)
ASMI	5(16%)
ALMI	2(6%)
AWMI	6(20%)
IWMI + RWMI	3(10%)
IWMI + RWMI + PWMI	2(6%)

Table no14: Classification of ACS patients with troponin test

TROPONIN TEST	NO OF PATIENTS (n=50)
Positive result	38
Negative result	12

Table no 15: Classification of patients with no ST segment elevation with troponin test

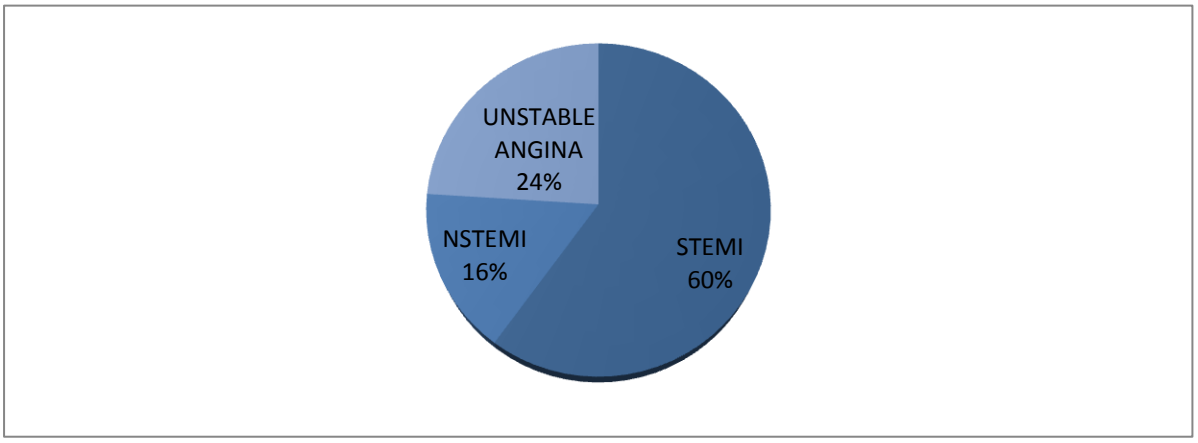
TROPONIN TEST	NO OF PATIENTS (n=20)
Positive result(NSTEMI)	8
Negative result(UNSTABLE ANGINA)	12(60%)

Out of 50 patients diagnosed with acute coronary syndrome, 60% had STEMI. IWMI was the leading finding in ECG of 40% patients with STEMI.

76% of the patients were Trop T positive, out of which 16% was contributed by NSTEMI patients.

Out of 30 STEMI patients, 25 were thrombolysed. The remaining 5 did not undergo thrombolysis due to various reasons.

Figure no 7 Pie chart distribution of ACS



KILLIP STAGING

Killip staging was done for patients with MI. 55.2% of the patients belonged to Killip class I.

Table no 16

KILLIP STAGING	NO OF PATIENTS(n=50)
Killip class I	21(42%)
Killip class II	6(12%)
Killip class III	6(12%)
Killip class IV	5(10%)

ANALYSIS OF RESULTS

This was a cross sectional study done on 50 patients who were admitted with acute coronary syndrome. Out of the 50, 78% (39) had presented with chest pain and 30.7% of these 39 patients gave the typical history of angina pain. 18% &20% gave the history of nausea and vomiting respectively. No chest pain was reported by 22% (110 of the study group. Out of these 11 patient 72.7% (8) were above the age of 60 years .82% of those without chest pain were known diabetics

The most common sign was crepitations or basal crackles (30%). 8 %(4) of the patients had hypotension while 10% had hypertension at the time of presentation. S3 was heard in 12% of the study group and 22% (11) had extensive crackles (pulmonary edema)

Most of the patients belongs to the age group of 45 to 50 years (54%) and 34% (17) belongs to the age group of 60 to 75 years 8% (4) were above the age of 75

In this study 32% (16) had diabetes out of which 285 (140 were diagnosed with diabetes >10years while 4% were diagnosed diabetes <10 years. 50% (25) of the study group were diagnosed as hypertensive patients

Out of 50 female ACS patients 88% (44) had attained menopause were 12% were in the pre menopausal age group.16 %(8) of the female patients gave appositive history

of OCP use on a regular basis. While one out of 50 gave a history of alcohol use and 18 %(9) gave a history of tobacco use.

50%(25) of the female patients were found to have sedentary life style 44% (22) and 6%(3)were found to be moderate and heavy workers respectively.6%(3)were gave a history of regular exercise and 8% gave a history of irregular exercise.4%(2) were found to have dietary planning. Majority of the patients i.e. 82 %(41) did not follow dietary planning nor exercise.

8 %(4) 14 %(7) and 6 %(3) gave past history of TIA, IHD and thyroid disorders respectively

Out of 50 patients 48%(24) were found to have a BMI of 25 – 29.9 and 36%(18)had a normal BMI OF 18.5 -24.9.10%95),2%(1)and 2%(1) belongs to class 1 ,class2 and class 3 obesity respectively.

48%(24) had an HDL 20-30 mg/dl and 34%(17) had an HDL of 30 -40 only 2%(1) had desirable HDL value of >50 mg/dl.40%(20) of the female ACS patients had an LDL level of >160 mg/dl.38%(19) and 22%(11)had a LDL value of <130 and 130 - 160 mg/dl respectively

Out of 50 patients I this study group 60%(30) had STEMI.40%(12) of the 30 STEMI patients had IWMI 20%(6) had AWMI 16% (5) 6(2)and6%(2) had ASMI ,ALMI,IWMI RVMI AND PWMI respectively.

76% of the patients were Trop t positive out of which 16% was contributed by NSTEMI.24% (12) of the 50 patients had unstable angina. Out of the 30 STEMI patients 25 were thrombolysed

According to Killip staging 42 %(21) belong to class 1 and 10% (5) belongs to class 4 the remaining 24% belongs to class 2 and 3 equally.

DISCUSSION

Acute coronary syndrome in females has become a real challenge for both physicians and cardiologists. Most of the data regarding the risk factors of ACS is based on studies conducted in men.

According to various studies done around the world cardiovascular disease is one of the leading causes of death in men and women. According to WHO report 55% deaths in European females is related to ACS as compared to 43% in men. However the mortality from CVD has declined in the developed countries from 1970 to 2015 while it has doubled in the developing countries.

In a study by V. Chiamvimonvat and L. Sternberg (University of Toronto):
Coronary artery disease is the leading cause of mortality in women, with incidence after menopause equal to that of men. Diabetes and postmenopausal status without Hormone replacement therapy is the strongest risk factors.

It has been reported that people of the South Asian descent has a higher incidence of coronary heart disease. Metabolic abnormalities like high TG concentration, type 2 DM, low HDL and central obesity are also more prevalent in the people from Indian subcontinent

For men and women cardiovascular risk increases with age, smoking, obesity, hypertension and hyperlipidemia. In our cross sectional study of 50 females with ACS, **54% belongs to the age group of 45 to 60 years**. The number of female patients above 75 years was only 8%, while 34% of study group belongs to 60 -75 age group. This discrepancy can be attributed to poor hospital reporting and increased mortality among the ACS female patients above 75 years.

The coronary risk factors in both sexes are the same but after menopause their influence on women is different. This is due to the female specific hormonal changes during menopausal period. The data collected from POLISH ACS registry revealed that women with ACS were older than men by 7.7 years**. In our study, 88% of the female patients had attained menopause and this was clearly indicative of the age related and hormonal influence on the incidence of ACS .**

In our study, 78% of the patients had chest pain where as 22% did not give a typical history of chest pain or angina; 38% of the study group had symptoms of nausea and vomiting and 20% had palpitations.

Out of the 11 patients without chest pain, 9 were diabetic. This observation is in accordance with various studies which showed the prevalence of atypical symptoms in diabetic individuals.

In a study by **Marrugat et al., (1998)** women are at a higher risk of having symptoms such as nausea, instead of chest pain.

Out of 50 patients, 32% were found to have crepitations, while 24% patients had pulmonary oedema or extensive crackles.

According to the CHINESE ACS registry, 24.4 % of women were significantly older than male patients and the prevalence of diabetes and hypertension were higher in the female group compared to the male group. Similar observations are seen in other worldwide registries also. In our cross sectional study of 50 patients, 16 were diabetic and out of which 14 patients were known diabetic for more than 10 years

Women with diabetes had a 3.5times increased cardiovascular related mortality as compared to non diabetic women according to Huxley et al.

Diabetes confers substantial increased relative risk of first incident and admission for MI in women compared with age-to-sex matched controls younger than age 65 yrs over a 20 year follow up in **Copenhagen City Heart Study**.

In our study 50% of the patients were hypertensives. Along with high salt intake, genetic factors, obesity and psychological stress plays an important role in the development of hypertension. Hypertensive patients had an increased morbidity and mortality following a cardiovascular incident.

An interesting finding is that women smokers have a six fold increased risk of ACS compared to a non-smoker female, while smoking men only had a threefold risk increase. This can be attributed to the effect of nicotine on the oestrogen level. In our

study of 50 patients, 2 gave a positive history of smoking and 7 gave a history of tobacco chewing.

A study by Liaquat Ali Cheema et al., (Gender comparison of coronary risk factors and clinical presentation in Pakistani patients with coronary artery lesions): This showed that smoking was not a risk factor in females in the study population and diabetes mellitus was more common in females while smoking and dyslipidemia in males. However this can be attributed to cultural and social differences also.

Mosca et al study: In this study, compared with male patients, out of the total female patients, 41% were identified to be overweight, 36% said smoking, 31% cited high cholesterol, 29% identified family history, 19% said hypertension, 19% identified diabetes and 1% had high triglyceride level (Mosca et al., 2004).

In our study 48% were overweight and 10% belongs to class 1 obesity and 36% of the patients had a normal BMI. The importance of activities in the prevention of cardiovascular disease is evident in this study group.

In our study, 50% of the female patients led a sedentary life style (activities based on occupation) while 3 out of the fifty people were heavy workers. 82% of the female patients did not follow any life style modification and 2 out of the 50 patients had dietary planning and regular exercise.

In the *Nurses' Health Study*, both BMI >25 and physical activity were important predictors of coronary artery disease in 20 year follow up.

In men, high LDL levels were found to have a strong prognostic impact where as in women the role of low HDL is more important. Independent of the serum LDL and TG levels, women with low HDL levels had a higher risk for CVD. The risk of CAD decreases by 3% for every 1 mg/dl increase in HDL in women but only 2% in men.

In this study, 20 (40%) out of the 50 patients had an LDL above 160, while favourable lipid profile was seen only in 19 patients. 50% of the study group had HDL values below 30.

In its recent guidelines, the American Heart Association(AHA) has changed the cut-point for what constitutes low HDL-C in women from less than 40 to less than 50.

Recently published data on mortality from coronary heart disease have shown an increase in the mortality of women between 35 &45 years. Also coincident with this observation, there is more frequent use of OCPs among this age group. In our study of fifty patients, 8 females had a history of OCP use.

In this study, menopause was found to be the most prevalent risk factor (i.e. 88% of the total) followed by dyslipidemia. A low HDL value (i.e. 50% of the total) was found to be more prevalent than a high LDL value. The next important risk factor according to this study was a deranged body mass index. This was followed by hypertension, diabetes and tobacco use.

CONCLUSION

The following are conclusions that could be inferred from this study on clinical spectrum and risk factors among female patients.

- The most significant risk factor was age, followed by dyslipidemia and abnormal BMI.
- The most common age group affected was 45-60 years
- Most of the female patients had attained menopause. This highlights the importance of hormonal influence on ACS.
- Most of the patients had a low HDL value below 30mg/dl and LDL above 160mg/dl
- Diabetes and hypertension are clearly associated with an increased risk for ACS.
- Obesity, sedentary lifestyle and lack of exercise are also important risk factors for ACS
- Tobacco and OCP use also can be considered as risk factors in the development of ACS.
- The most common symptom was chest pain. Atypical symptoms were more prevalent among diabetics and the elderly
- Most of the patients had STEMI, followed by unstable angina.

BILIOGRAPHY

1. Ishihara M , Kojimia S , Sakemato T , Ashada Y , Kinura K et al ;Usefulness Of Combined White Blood Cell Count and Plasma Glucose For Predicting In-Hospital outcomes After Acute Myocardial Infarction . American journal of Cardiol 2006;97:1558-1563.

2. Harrison's Principles Of Internal Medicine volume -2; .18th edn page no:1448-1458.

3. Antman EM,Braunwald ;ST segment elevation Myocardial infarction.In:Zipes, Libby, Bonow, Braunwald editors.Braunwald's Heart disease a textbook of cardiovascular medicine. 7thedn. Philadelphia:Elsiever Saunders;2005.page n0:1141-1142

4. Arthur.C.Guyton, John.E.Hall. Resistance of body to infection -1 In Guyton Textbook of physiology.10th edition.Philadelphia: Elsiever Saunders;2006.p392.

5. Chandler AB, Chapmann I, Erihadt LR, Roberts WC, Schwartz CJ, Sinapius D, et al. Coronary thrombosis in myocardial infarction. Report of a workshop on the role of coronary thrombosis in the pathogenesis of acute myocardial infarction. Am J Cardiol 1974; 34: 823–833.

6. R. Ross, Atherosclerosis—an inflammatory disease.N Engl J Med 1999; 340:115– 126.

7. Amaro, J.R. Gonzalez-Juanatey and C Iglesias et al., Leukocyte count as a predictor of the severity ischaemic heart disease as evaluated by coronary angiography .Rev Port Cardiol 1993;12:913–917

8. Weitman M, Cook S, Auinger P et al. Tobacco smoke exposure is associated with the metabolic syndrome in adolescents.Circulation 2005;112:862-869.

9.Schneider JG, Tompkins C, Blumenthal RS, Mora S. The metabolic syndrome in women. Cardiol Rev 2006;14;286-291.

10. Wlid R, Polycystic ovary syndrome: A risk for coronary artery disease? Am J Obstet Gynecol

2002;186 (1):35-43.

11. Giannitsis E, Katus HA: *Biomarkers of necrosis for risk assessment and management of STelevation myocardial infarction*. In: Morrow DA, ed. *Cardiovascular Biomarkers: Pathophysiology and Disease Management*, Totowa, NJ: Humana Press; 2006:119-128.

12. Apple FS, Quist HE, Doyle PJ, et al: Plasma 99th percentile reference limits for cardiac Troponin and creatine kinase MB mass for use with European Society of Cardiology/American College of Cardiology consensus recommendations. *Clin Chem* 2003; 49:1331.

13. Jaffe AS, Babuin L, Apple FS: Biomarkers in acute cardiac disease: The present and the future. *J Am Coll Cardiol* 2006; 48:1.

14. Sabatine MS, Morrow DA, Giugliano RP, et al: Association of hemoglobin levels with clinical outcomes in acute coronary syndromes. *Circulation* 2005 ; 111:2042.

15. Moon JC, De Arenaza DP, Elkington AG, et al: The pathologic basis of Q-wave and non-Qwave myocardial infarction: A cardiovascular magnetic resonance study. *J Am Coll Cardiol* 2004; 44:554.

16. Cheitlin MD, Armstrong WF, Aurigemma GP, et al: ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

17. Rosenberg RD, Aird WC: Vascular-bed–specific hemostasis and hypercoagulable states. *N Engl J Med* 1999; 340:1555.

18. Argulian E, Patel AD, Abramson JL, et al. Gender differences in short-term cardiovascular outcomes after percutaneous coronary interventions. Am I Cardiol 2006;98(1):48-53.

19. Haan CK, Chiong JR, Coombs LP, et al. Comparison of risk profiles and outcomes in women versus men >75 years of age undergoing coronary artery bypass grafting. Am J Cardiol 2003;91:1255-1258.

20. Albert CM, Chae CU, Grodstein F, et al. Prospective study of sudden cardiac death among women in the United States. Circulation 2003;107(16):2096-2101.

21. De Bacquer D, De Backer G, Kornitzer. Prevalence of ECG findings in large population based samples of men and women. Heart 2000;84:625-633.

22. Tunstall-Pedoe H, Kuulasmaa K, Amouyel P, et al: Myocardial infarction and coronary deaths in the World Health Organization MONICA Project. Registration procedures, event rates, and case-fatality rates in 38 populations from 21 countries in four continents. *Circulation* 1994; 90:583

23 Mosca L, Linfante A, Benjamin E, et al. National Study of Physician Awareness and Adherence to Cardiovascular Disease Prevention Guidelines. Circulation 2005; 111; 499-510.

24. Mosca L, Mochari H, Christian A , et al. National Study of women's Awareness, Preventive Action, Barriers to Cardiovascular Health. Circulation2006; 113; 525-534.

25. The Women's Caucus, Working Group On Women's Health of the Society of General Internal Medicine. Hypertension in Woman : what is really known? Ann Intern Med 1991; 115: 287-293

26. Christou D, Jones P, Jordan J, Diedrich A, Seals D. Women have lower tonic support of arterial blood pressure and less effective baroreflex buffering than men. Circulation 2005;111: 494-498.

27. Sagie A. The natural history of borderline isolated systolic hypertension. N Engl J Med 1993;329: 1912-1917.

28 Franklin SS Pio Jr, Wrong ND, et al. Predictors of new onset diastolic and systolic hypertension: Framingham Heart Study. Circulation 2005; 111:1121-1127.

29 Cooper WO, Hernandez-Diaz S, Arbogast PG. Major congenital malformations after first trimester exposure to ACE inhibitors. N Engl J Med 2006; 354: 2443-2451.

30. Schaefer BM, Caracciolo V, Frishman WH, Charney P. Gender, ethinicity and genetics in cardiovascular disease: part II. Implications for pharmacotherapy. Heart Dis 2003;5: 202-214.

31. Walsh JME, Pignone M. Drug treatment of hyperlipiudemia in women. JAMA 2004;291: 2243-2252.

32. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1994-2004. JAMA 2004; 295: 1549-1555.

33. Rennnert NJ, Charney P. Preventing cardiovascular disease in diabetes and glucose intolerance: evidence and implications for care. Prim Care 2003;30: 569-592.

34. Whitley L, Padmanabhan S, Hole D, Isles C. Should diabetes be considered a coronary artery disease equivalent? Results from 25 years of follow up in the Renfrew and Paisley Survey. Diabetes Care 2005; 28: 1588-1594.

35. Almdal T, Scharling H, Jensen J, Vestgaard H. The independence effect of type 2 diabetes

on ischemic heart disease, stroke and death. Arch Intern Med 2004; 164 :1422-1426

36. Barrett-Conner E, Giardina E, Gitt U, Tschoepe D. Women and heart disease:the role of diabetes and hyperglycemia. Arch Intern Med 2004;164:934-942.

37. Vaccarino V, Parsons L, Every N, et al. Impact of history of diabetes on hospital mortality in men and women with first acute myocardial infarction. Am J cardiol 2000;85:1486-1489.

38. Persell S, Baker D. Aspirin use among adults with diabetes. Arch Intern Med 2004;164:2492-2499.

39. Prospective Diabetes Study Group. United Kingdom Prospective Diabetes Study 27: Plasma lipids and lipoproteins at diagnosis of NIDDM by age and sex,UK:Diabetes Care 1997;20(11):1683-1687.

40. Expert Panel on Detection, Evaluation, and treatment of High Blood Cholesterol in Adults (adult Treatment Panel III).JAMA 2001;285:2486-2497.

41. Weitman M, Cook S, Auinger P et al. Tobacco smoke exposure is associated with the metabolic syndrome in adolescents.Circulation 2005;112:862-869.

42. Schneider JG, Tompkins C, Blumenthal RS, Mora S. The metabolic syndrome in women. Cardiol Rev 2006;14;286-291.

43. Wlid R, Polycystic ovary syndrome: A risk for coronary artery disease? Am J Obstet Gynecol 2002;186 (1):35-43.

44. Aoridonize T, Essah P, Iuorno M, Nestler J. Prevalence and characteristics of the metabolic syndrome in women with polycystic ovary syndrome. J clin Endocrinol Metab 2005;90(4):1929-

1935.

45. Winkleby MA, Robinson TN, Sundquist J, Kraemer HC. Ethnic variations in cardiovascular disease risk factors among children and young adults: findings from the third National Health

and Nutrition Examination Survey,1988-1994.JAMA 1999;281:1006-1013.

46. Wilson PE, Sullivan L, Kannel WB. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. Arch Intern Med 2002;162:1867-1872.

47. Yanoviski SZ, Yanoviski JA. Obesity. N Engl J Med 2002;346:591-602.

48. Charney P ed. Coronary artery disease in Women: prevention, Diagnosis and Management. Philadelphia: American College of Physicians:1999.

49. Women's Health Initiative Steering Committee. Effects of conjugated equine estrogen on postmenopausal women and hysterectomy: Women's Health Initiative randomized controlled trial. JAMA 2004;291:1701-1712.

50. Sharkey SW, Lesser JR, Zenovich AG, et al. Acute and reversible cardiomyopathy provoked by stress in women from the United States. Circulation 2005;111(4):472-479.

51.Argulian E, Patel AD, Abramson JL, et al. Gender differences in short-term cardiovascular outcomes after percutaneous coronary interventions. Am I Cardiol 2006;98(1):48-53.

52. Haan CK, Chiong JR, Coombs LP, et al. Comparison of risk profiles and outcomes in women versus men >75 years of age undergoing coronary artery bypass grafting. Am J Cardiol

2003;91:1255-1258.

PROFORMA

NAME

AGE

DOA

DOD

MARITAL STATUS **NO OF CHILDREN**

ADDRESS

OCCUPATION

FAMILY INCOME/YEAR

PRESENTING SYMPTOM: Chest pain / Nausea / vomiting / Epigastric pain / Jaw pain / Left arm pain / Sweating/ Dyspnoea / Palpitation / Syncope / Others

DURATION OF SYMPTOMS :

PAST HISTORY :

- PAST H/O IHD
- TIA/STROKE
- HYPERTENSION: YES/ NO TREATMENT
- DIABETES: YES/ NO TREATMENT
- HYPERLIPIDEMIA: YES / NO TREATMENT
- THYROID DISORDERS : YES/ NO
- RHEUMATOLOGICAL / CONNECTIVE TISSUE DISORDERS:
- SYPHILIS:
- SMOKING: YES/NO
- ALCOHOL:
- CHRONIC DRUG INTAKE / OCP:

- PHYSICAL ACTIVITY:

FAMILY HISTORY OF IHD:

MENSTRUAL HISTORY:

MENOPAUSE: YRS SINCE MENOPAUSE

DIET HISTORY: VEG/NON VEG

TYPE OF COOKING OIL:

SALT INTAKE:

COFFEE/ TEA:

CLINICAL EXAMINATION

HT

WT

BMI

BP:

PR:

Anemia:

Cyanosis:

Clubbing:

Icterus:

Pedal edema:

Markers of Hypercholesterolemia

CVS:

RS:

P /A:

CNS:

LABORATORY INVESTIGATIONS

1. Urine R /E:

Albumin

Sugar

Deposits

2. RFT: Urea

Creatinine

Electrolytes

3. CBC: Total Count Differential Count

Erythrocyte Sedimentation Rate

Hemoglobin Platelets

4. RBS

5. LIPID PROFILE

TOTAL CHOLESTEROL mg/dl

HDL CHOLESTEROL mg/dl

LDL CHOLESTEROL mg/dl

VLDL CHOLESTEROL mg/dl

TRIGLYCERIDE mg/dl

6. Serum Troponin -T:

7. Electrocardiogram:

8. Echocardiogram:

OTHER RELEVANT INVESTIGATIONS

DIAGNOSIS

THROMBOLYSIS - DONE/NOT DONE

IN HOSPITAL COMPLICATIONS

LIST OF ABBREVIATIONS

ACS – Acute coronary syndrome

AMI– Acute myocardial infarction

AF– Atrial Fibrillation

CAD – Coronary Artery Disease

CCF – Congestive Cardiac Failure

CHD – Coronary Heart Disease

CK-MB – Creatinine Kinase-MB

CVD – Cardiovascular diseases

ECG – Electrocardiogram

IHD – Ischemic Heart Disease

LVF – Left ventricular Failure

MR – Mitral Regurgitation

MI – Myocardial infarction

RBS – Random Blood Sugar

SA– Sinoatrial node

STEMI – ST Elevation myocardial infarction

VT – Ventricular Tachycardia

WBC – White Blood Cell

Sl no	IP no	age	**symptoms**					DM		HTN	PRIOR IHD	TIA/ STROKE	RHD/ SLE	THYROID	Menopause, OCP use
			CP	EP	pal	dys	oth	<10 YRS	>10 YRS						
1	3980	53	N	N	Y	N	N	N	Y	N	N	N	N	N	M,ocp
2	4011	57	Y	N	N	Y	Y	Y	N	N	N	N	N	Y	M,nil
3	4078	76	N	N	Y	Y	Y	N	Y	Y	N	Y	N	N	M,nil
4	4678	62	Y	Y	N	Y	N	N	N	N	N	N	N	N	M,nil
5	5789	58	Y	N	Y	N	N	N	N	Y	Y	N	N	N	M,nil
6	5999	65	Y	N	N	Y	Y	N	N	N	N	N	N	N	M,nil
7	7120	52	Y	N	N	N	N	N	N	Y	N	N	N	N	M,nil
8	7130	53	Y	Y	N	N	N	N	N	N	N	N	N	N	M,nil
9	7390	66	N	N	Y	Y	N	N	Y	Y	Y	N	N	N	M,nil
10	7780	43	Y	N	N	N	N	Y	N	N	N	Y	N	N	-,ocp
11	7990	55	Y	N	N	N	N	N	N	N	N	N	N	N	M,nil
12	8000	77	N	N	Y	Y	Y	N	Y	N	N	N	N	N	M,nil
13	8012	59	Y	Y	N	Y	N	N	N	Y	N	N	N	N	M,nil
14	8027	58	Y	N	N	N	N	N	N	Y	N	N	N	N	M,nil
15	8456	62	Y	N	N	N	N	N	N	N	N	N	N	N	M,nil
16	8678	49	N	N	Y	Y	Y	N	Y	Y	N	N	N	N	M,nil
17	8654	72	Y	Y	N	N	N	N	N	N	Y	N	N	N	M,nil
18	3456	53	Y	N	N	N	Y	N	Y	N	N	N	N	Y	M,nil
19	8790	56	Y	Y	N	Y	N	N	N	Y	N	N	N	N	M,nil
20	8890	47	Y	N	N	N	Y	N	N	N	N	N	N	N	-,ocp
21	4567	59	Y	N	N	N	Y	N	N	Y	N	N	N	N	M,nil
22	4890	66	Y	Y	N	Y	N	N	N	Y	N	N	N	N	M,nil
23	9000	54	Y	N	N	N	Y	N	Y	N	Y	N	N	N	M,nil
24	9044	42	Y	N	N	Y	N	N	N	Y	N	N	N	N	-,nil
25	9051	72	Y	N	N	N	Y	N	N	N	N	N	N	N	M,nil
26	9160	76	N	N	N	N	N	N	Y	N	N	N	N	N	M,nil

27	9340	57	Y	N	N	N	N	N	Y	Y	N	N	N	N	M,ocp
28	9567	68	Y	N	N	N	Y	N	Y	Y	N	N	N	N	M,nil
29	9890	64	Y	N	N	N	N	N	Y	N	N	N	N	N	M,nil
30	10012	70	N	N	N	N	N	N	y	Y	N	N	N	N	M,nil
31	10056	59	Y	Y	N	N	Y	N	N	N	N	N	N	N	M,nil
32	5342	67	N	N	Y	Y	N	N	N	Y	N	N	N	N	M,nil
33	5129	59	Y	N	N	Y	Y	N	N	Y	N	N	N	N	M,nil
34	10067	60	Y	N	N	N	N	N	N	Y	N	N	N	N	M,ocp
35	10789	48	Y	Y	N	Y	Y	N	N	N	N	N	N	N	M,nil
36	10986	55	Y	N	N	N	N	N	N	N	N	N	N	N	M,nil
37	11198	73	Y	N	N	Y	N	N	N	Y	Y	N	N	N	M,nil
38	9410	50	Y	Y	N	N	Y	N	N	Y	N	N	N	N	M,nil
39	11567	52	Y	Y	N	Y	Y	N	N	N	N	N	N	Y	M,nil
40	14567	65	Y	N	N	N	N	N	N	Y	N	N	N	N	M,nil
41	15378	53	Y	N	N	N	N	N	N	Y	N	N	N	N	M,nil
42	15563	48	Y	N	N	N	Y	N	N	N	N	N	N	N	-,ocp
43	15768	79	N	N	Y	Y	N	N	Y	Y	Y	Y	N	N	M,nil
44	15890	63	Y	N	N	Y	Y	N	N	N	N	N	N	N	M,ocp
45	15967	58	Y	N	N	N	N	N	N	Y	N	N	N	N	M,nil
46	15980	73	N	N	N	N	N	N	N	Y	Y	N	N	N	M,nil
47	16000	59	N	N	N	Y	N	N	Y	N	N	N	N	N	M,nil
48	16008	52	Y	N	N	N	N	N	N	Y	N	N	N	N	M,nil
49	16120	49	Y	N	N	Y	Y	N	N	N	N	N	N	N	-,ocp
50	16759	68	Y	N	N	N	N	N	N	N	N	Y	N	N	M,nil

Sl.no	ALC/SMO	BMI	SIGNS								HDL	LDL	RBS	ECG	TROP T	Thrombolyse	
			Hyper T	Hypo T.	S3	S4	JVP	Creps	PE	KILLIP							physical activity
1	N	21	N	Y	Y	N	N	Y	Y	IV	42	170	112	IWMI	+	Y	sedentary
2	N	25.5	Y	N	N	N	N	N	N	I	52	180	100	ALMI	+	Y	sedentary
3	N	20.6	N	N	N	N	N	N	Y	III	38	109	176	AS MI	+	N	moderate
4	N	24	Y	N	N	N	N	N	N	I	27	180	167	AWMI	+	Y	sedentary
5	N	26	N	Y	N	N	N	Y	Y	IV	36	169	140	AWMI	+	N	sedentary
6	TOB	27.6	N	N	N	N	Y	Y	Y	III	49	172	146	IW+RWMI	+	Y	sedentary
7	N	33.6	N	N	N	N	N	N	N	II	34	110	112	IWI	+		Moderate
8	N	34.1	N	N	N	N	N	N	N		24	164	166	IWI	-		heavy
9	N	38.1	N	Y	N	N	Y	Y	Y	IV	44	119	157	IW+RWMI	+	N	sedentary
10	N	25.4	N	N	N	N	N	Y	Y	III	38	180	116	AWMI	+	Y	moderate
11	N	29	Y	N	N	N	N	N	N	I	47	178	80	ALMI	+	Y	moderate
12	N	25.2	N	N	N	N	N	N	N	I	35	172	244	IWMI	+	Y	sedentary
13	N	26.6	N	N	N	N	N	Y	N	II	42	182	95	AWMI	+	Y	moderate
14	N	28.7	N	N	N	N	N	N	N		32	189	89	IWI	-		sedentary
15	TOB	34	Y	N	N	N	N	Y	N	I	38	192	134	IWI	+		sedentary
16	N	25	N	N	N	N	N	Y	Y	IV	46	176	224	IWMI	+	Y	sedentary
17	N	22	N	N	Y	N	N	Y	Y	III	47	189	226	IWMI	+	N	moderate
18	TOB	26.1	N	N	N	N	N	N	N		33	172	119	AWI	-		sedentary
19	N	31	Y	N	N	N	N	N	N	I	36	194	212	IWMI	+	Y	sedentary
20	N	25.2	N	N	N	N	N	Y	N	II	48	179	134	AWMI	+	Y	sedentary
21	N	41	N	N	N	N	N	N	N		34	116	117	AWI	-		moderate
22	SMOK	27	N	N	N	N	N	N	N	I	28	180	116	IWMI	+	Y	sedentary
23	N	26.2	N	N	N	N	N	N	N		36	120	108	AWI	-		sedentary
24	N	26.7	N	N	N	N	N	N	N	I	31	119	226	AWI	+		sedentary
25	TOB	19.8	N	N	N	N	N	N	N		44	100	118	AWI	-		moderate
26	N	21	N	N	N	N	N	N	N		37	88	150	AWI	-		moderate
27	N	20.8	N	N	N	N	N	N	N		26	156	80	IWI	-		sedentary

28	N	25.7	N	N	Y	N	N	Y	N	II	39	110	200	IWMI	+	Y	moderate
29	N	27.7	N	N	N	N	N	N	N	I	44	144	86	ASMI	+	Y	sedentary
30	N	27	N	Y	N	N	N	N	Y	IV	37	176	188	ASMI	+	Y	sedentary
31	ALC	20	N	N	Y	N	N	Y	Y	III	49	177	250	AWI	+		sedentary
32	SMOK	25.6	N	N	N	N	N	N	N	I	33	174	100	IWMI	+	Y	moderate
33	N	22	N	N	N	N	N	Y	N	II	42	112	85	IWI	-		moderate
34	N	26	N	N	N	N	N	N	N	I	38	165	207	RW+PW+IW	+	Y	moderate
35	TOB	29	N	N	Y	N	Y	Y	Y	III	27	142	170	IW+RWMI	+	Y	moderate
36	N	27.2	N	N	N	N	N	N	N		36	137	117	AWI	-		heavy
37	N	24.8	N	N	N	N	N	N	N	I	38	158	109	IWMI	+	Y	moderate
38	TOB	21	N	N	N	N	N	N	N	I	39	119	120	IWMI	+	Y	moderate
39	N	19.4	N	N	N	N	N	N	N	I	35	174	160	AWMI	+	Y	moderate
40	N	25	N	N	N	N	N	N	N	I	32	177	152	AWI	+		sedentary
41	N	24.5	N	N	N	N	N	N	N	I	28	139	121	IWI	+		sedentary
42	TOB	32	N	N	N	N	N	N	N		47	104	92	IWI	-		sedentary
43	N	20.1	N	N	N	N	N	N	N		36	139	208	AWI	-		heavy
44	N	24	N	N	N	N	N	N	N	I	33	179	230	ASMI	+	Y	moderate
45	N	25.2	N	N	N	N	N	N	N	I	39	179	137	AWI	+		sedentary
46	SMOK	21	N	N	N	N	Y	N	N	I	43	118	200	IWMI	+	N	moderate
47	N	26.3	N	N	N	N	N	N	N	I	42	167	156	IWI	+		moderate
48	N	19.7	N	N	Y	N	N	Y	N	II	36	176	87	ASMI	+	Y	sedentary
49	N	22	N	N	N	N	N	N	N	I	31	111	100	IWMI	+	Y	sedentary
50	N	18.4	N	N	N	N	Y	N	N	I	47	118	80	PW+RW+IW	+	Y	moderate

KEY to MASTER CHART

Y = yes

N = no

CP= chest pain

EP= epigastric pain

Pal = palpitation

Dys = dyspnoea

OCP = oral contraceptive pill

M = menopause

ALC = alcohol

Smok = smoking

Tob = tobacco chewing

ASMI = anteroseptal mi

AWMI= anterior wall mi

IWMI = inferior wall mi

ALMI = anterolateral mi

RVMI= right ventricular mi

PWMI = posterior wall mi

PE =pulmonary oedema

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR SOBIN E.JOSEPH** Post graduate in the Department of General Medicine ,Thanjavur Medical College & Hospital, Thanjavur – 613004 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations

Place :

Date : Signature of participant

INFORMATION SHEET

We are conducting a cross sectional study on **THE PREVALENT RISK FACTORS OF ACS IN FEMALES**

in the Department of General Medicine , Thanjavur Medical College & Hospital, Thanjavur – 613004.

- At the time of announcing the results and suggestions, name and identity of the patients will be confidential.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator **Signature of participant**

Date:

KEY WORDS

ACS – Acute coronary syndrome

AMI– Acute myocardial infarction

AF– Atrial Fibrillation

UA – unstable angina

CAD – Coronary Artery Disease

CCF – Congestive Cardiac Failure

CHD – Coronary Heart Disease

CK-MB – Creatinine Kinase-MB

CVD – Cardiovascular diseases

ECG – Electrocardiogram

IHD – Ischemic Heart Disease

LVF – Left ventricular Failure

MR – Mitral Regurgitation

MI – Myocardial infarction

RBS – Random Blood Sugar

SA– Sinoatrial node

STEMI – ST Elevation myocardial infarction

VT – Ventricular Tachycardia

WBC – White Blood Cell